A Dissertation on

"CLINICAL AND ETIOLOGICAL PROFILE OF EPILEPSY WITH ONSET WITHIN THE FIRST 3 YEARS OF LIFE IN A TERTIARY CARE HOSPITAL"

Submitted to the

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirement for the award of degree of

DM
BRANCH-I

NEUROLOGY

DEPARTMENT OF NEUROLOGY

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

AUGUST 2013

CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL AND ETIOLOGICAL PROFILE OF EPILEPSY WITH ONSET WITHIN THE FIRST THREE YEARS OF LIFE IN A TERTIARY CARE HOSPITAL"** is a bonafide original research work done by **Dr. A. RAJENDRAN,** in partial fulfilment of the requirement for D.M., Branch-I, Neurology examination of The Tamilnadu Dr. M.G.R. Medical University to be held in AUGUST 2013, under the direct supervision and guidance of **PROF. Dr. S.GOBINATHAN, M.D., D.M (Neurology).,** Professor and Head, Department of Neurology at Stanley Medical College and Hospital, Chennai.

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DECLARATION

I, **Dr. A. RAJENDRAN,** Solemnly declare that the dissertation titled **"CLINICAL AND ETIOLOGICAL PROFILE OF EPILEPSY WITH ONSET WITHIN THE FIRST THREE YEARS OF LIFE IN A TERTIARY CARE HOSPITAL"** is a bonafide work done by me during the period of February 2012 to January 2013 at Government Stanley Medical College and Hospital, Chennai, under the guidance and supervision of **PROF. Dr. S.GOBINATHAN, M.D., D.M (Neurology).,** Professor and Head, Department of Neurology, Government Stanley Medical College and Hospital, Chennai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the rules and regulations for the award of **D.M Degree, Branch-I, Neurology** examinations to be held in August-2013.

Place: Chennai **(Dr. A. RAJENDRAN)**

Date:

ACKNOWLEDGEMENTS

I wish to express my sincere thanks to **Prof. Dr. S. GEETHA LAKSHMI, MD., Ph. D.,** Dean, Government Stanley Medical College and Hospital for having permitted me to utilize the facilities of the hospital for the conduct of the study.

My heartfelt gratitude to our beloved chief **Prof. Dr.S.GOBINATHAN, M.D., D.M (Neurology).,** Professor and Head, Department of Neurology, Government Stanley Medical College and Hospital for his guidance, motivation, valuable suggestions, expert supervision and for making all necessary arrangements for conducting this study.

I am greatly indebted to **Prof. Dr. S. VELUSAMY, M.D., D.M (Neurology).,** Professor and Head, Department of Paediatric Neurology, Government Stanley Medical College and Hospital, for his constant encouragement and support and allowing me to conduct the study at the Department of Paediatric Neurology.

I am greatly indebted to **Prof. Dr. C.AMARNATH, M.D (Radio diagnosis)., FRCR., MNAMS.,** Professor and head, Department of Radiology, Government Stanley Medical College and Hospital, who offered guidance and radiological diagnosis throughout the period of the study.

I express my sincere gratitude to my Assistant Professor **Dr. MALCOLM JEYARAJ MD., D.M (Neurology)., PDF(Epilepsy).,** who had evinced constant and keen interest in the progress of my study right from the inception till the very end and was instrumental in the successful completion of the study.

I sincerely thank **Dr. S. SAKTHIVELAYUDAM, MD., D.M (Neurology).,** my Assistant Professor, for the help, keen interest and suggestions throughout the period of the study.

I sincerely thank **Dr. P. R. SOWMINI, MD., D.M (Neurology).,** my Assistant Professor, for the help, support and suggestions throughout the period of the study.

I sincerely thank **Dr. B. SUHASHINI, M.D (Radio diagnosis)., FRCR.,** Assistant Professor, Department of Radiology, Government Stanley Medical College and Hospital, for giving the radiological diagnosis and suggestions throughout the period of the study.

I thank **Dr. GANGADEVI, M.D (Radio diagnosis)., DMRD.,** and **Dr. K. SHIVA SHANKAR, DMRD, DNB.,** Assistant Professors, Department of Radiology, Government Stanley Medical College and Hospital, for their help throughout the period of the study.

My sincere thanks to all those **post graduates** who helped me during this study period.

I thank **Mr. A. ALBERT JOSEPH, M. Sc., DHS., PGDGA**., Statistician Schizophrenia Research Foundation(India) for helping me in statistical analysis.

I thank **Mr. RAVICHANDRASEKAR**, Electroencephalography technician for his help to record electroencephalography for our study population.

I thank the **Staff nurses and M.R.I Technicians**, Government Stanley Medical College and Hospital for their cooperation and assistance.

I owe my gratitude to **all the patients and their family** included in the study, for their whole hearted co-operation, without their cooperation this study would not have been possible.

CONTENTS

ABBREVIATIONS

AED	- Antiepileptic drug
BC	- Before Christ
CPS	- Complex partial seizure
CPS – ET	- Complex partial seizure of extra temporal origin
CPS-T	- Complex partial seizure of temporal origin
DALY	- Disability adjusted life years
EEG	- Electroencephalography
FI	- Focal cortical infarct
GNI	- Gross national income
GTCS	- Generalized tonic clonic seizure
HIE	- Hypoxic ischemic encephalopathy
ILAE	- International League Against Epilepsy
INR	- Indian National Rupees
LSCS	- Lower segment caesarian section
MRI	- Magnetic resonance imaging
NHBI	- Neonatal hypoglycemic brain injuries
PEC	- Perinatal encephaloclastic conditions
PVL	- Periventricular leucomalacia
SE	- Status epilepticus
SPS	- Simple partial seizure
SSC	- Semiological seizure classification
WHO	- World Health Organization
YLL	- Years of life loss

INTRODUCTION

INTRODUCTION

Epilepsy is a common neurological disorder. It affects nearly 50 million people worldwide without any national, geographical, ethnical, age and sex boundaries. The disease burden of epilepsy is 1 percent and it causes 6.4 million disability-adjusted life years (DALYs) worldwide and it causes 1.32 million years of life (YLL) loss.[1] Almost 80 percent people with epilepsy living in developing country including India. As of now, 6 to 10 million people are suffering from epilepsy in India.[2] Epilepsy is one of cost intensive disorder. It causes huge burden to the individuals, health care providers and society at large.[3]

The first year of human life is associated with the highest incidence of epilepsy.[4] During infancy a unique interface exists between epilepsy and normal brain maturation.[5] The causes of remote symptomatic seizure, occurring in early childhood are different from adults, it also differs in developing countries like India comparing to developed countries.[5] Only very few studies are available from India and no such study is available from this part of the country. So it is important to know the clinical and etiological profile of epilepsy in our children, which will help in adopting effective and better strategies for

epilepsy management and prevention or modifications of various factors relating to epilepsies.

Historical perspectives of epilepsy:

The word "Epilepsy" originated from Greek word 'Epilepsia' which means to seize, to take hold of or to attack. The word 'Seizure' originated from Latin word 'sacire' and means to claim. This particular description of epilepsy is actually reflecting the very nature of ancient faith that the people with epilepsy has been claimed or seized by supernatural power, god or sprit, mostly evil.[6] Epilepsy also referred as 'the falling sickness'. The ancient Sumerian term 'antasubba' and the later Assyrian and Babylonian word 'miqtu' are referring to 'the falling sickness'.[7,8] The following are the important ancient literatures about epilepsy: 1) Carakasamhitas of Ayurveda (1000-800 BC) 2) Agasthiyar kirigai nool in Tamil culture (6^{th} or 7^{th} century BC) 3) Babylonian clay tablet in the British Museum (2nd millennium BC) 4) The Hippocrates' treaties on 'On the sacred disease' (5^{th} century BC).

Ayurveda means knowledge of life (in Sanskrit ayu means life, and veda means to know). Original descriptions of Vedas are not available but most of its contents available today are through the Samhitas (the encyclopedic works) Caraka and Sushrutha. In

Carakasamhita (1000-800 BC), epilepsy has been mentioned as Apasmara or Apasmrti. Epilepsy has also been described as one of the 8 diseases known (diagnosed) by man that can be controlled with medical therapy. The 8th chapter (Nidanasthana- diagnosis) and 10th chapter (Chikitsasthana- treatment) of Carakasamhita are devoted exclusively to epilepsy.[9,10]

The word 'Siddhi' means achievement and siddhars were people who achieved certain results in medicine, tapas and yoga. The Siddha system of medicine was believed to be given by Lord Shiva to his wife Parvathi. This was subsequently handed down to Nandhideva who in turn gave this to siddhars. The origin of Tamil language and Siddha system of medicine were attributed to Sage Agasthiya, who was one of the 18 siddhars. He probably lived during 600 or 700 BC. There are many medical books ascribed to him, one among them is 'Agasthiyar kirigai Nool'. The Siddha system describes 5 major types of epilepsy which are Amarakandam, bhramakandam, kumarakandam, kakai vali and muyal vali.[11]

Babylonian clay tablet available at the British Museum (2nd millennium BC) is one of the 40 tablets of a Babylonian textbook of medicine (Sakikku). It is written in Neo-Babylonian script and is one of the oldest literatures describing epilepsy in detail (in number 26

cuneiform text). The Babylonian concept of epilepsy was that the manifestations of epilepsy were the work of demons and ghost. Greeks view on epilepsy were very similar to the Babylonian views. The Hippocratic corpus comprises roughly about 70 Hippocratic texts which contained the teaching of Hippocrates- the 'Father of medicine. The most influential part of the corpus is 'On the Sacred Disease'. In this book, Hippocrates confronted the popular view about the epilepsy. He had a revolutionary view that epilepsy is disease of brain and advised physical treatment. However, his view did not find its place till 18th and 19th century. The great Greek philosopher Socrates (469-399 BC) probably had temporal lobe epilepsy.[12]

Seizures and epilepsy-definitions of terminology;

Seizures: An epileptic seizure is defined as "a transient occurrence of symptoms and / or signs due to abnormal excessive or synchronous neuronal activity in the brain".[13]

Epilepsy: Epilepsy is defined as a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures and by the neurobiological, cognitive, psychological and social consequences of this condition. The diagnosis of epilepsy requires the occurrence of at least 1 unprovoked epileptic seizure with either a second such seizure or

enough electroencephalography (EEG) and clinical information to demonstrate an enduring predisposition to develop recurrences of seizures.[13,14,15] For epidemiological purposes, the diagnosis of epilepsy is made when 2 or more unprovoked epileptic seizures in a time frame of more than 24 hours.[13,14,15]

Symptomatic seizures: The definition of symptomatic epilepsy has undergone many a change over the years. Originally it was used to denote any epilepsy in which the cause was identified. Currently it is defined as follow "symptomatic epilepsy is epilepsy of an acquired or genetic cause, associated with neuroanatomical or neuropathological abnormalities indicative of an underlying condition or disease".[16]

Acute symptomatic seizure (provoked seizures, reactive seizures and situation related seizures)**:-** It is a clinical seizure occurring at the time of a systemic insult or in close temporal association with a documented brain insult. The time limits are suggested as follows, seizures occurring within 1week of anoxic encephalopathy, stroke, traumatic brain injury or intracranial surgery; at the identification of subdural hematoma; at the presence of active CNS infections; during active phase of multiple sclerosis or other autoimmune diseases. It is also diagnosed when seizures occur in the presence of severe metabolic derangements (which are documented within 24 hours by specific hematological and /or

biochemical abnormalities), alcohol or drug intoxication and its withdrawal, or exposure to epileptogenic drugs.[17]

Remote symptomatic epilepsy: - If the above epilepsy can be attributable to a preexisting, non-acute or static cause then it is referred to as remote symptomatic epilepsy. However there is a grey area in which the distinction between the acute and remote symptomatic epilepsy is rather arbitrary.[18]

Hypermotor seizures: These are seizures characterized by automatisms involving, predominantly, proximal limb or axial muscles and produce irregular sequential ballistic movements, such as pedaling, thrashing, pelvic thrusting movements.

Hypomotor seizures: Hypomotor seizures are characterized by arrest of behavioral motor activity or significant decrease of behavior motor activity with indeterminate level of consciousness.[19]

Automatisms: Automatisms are repetitive, patterned, semi purposeful motor activities. Gastaut described it as “more or less coordinated, adapted involuntary motor activity occur in association with altered sensorium either in the course of or after an epileptic seizure and usually with amnesia for the episode”.[20]

Aura: The term aura is derived from Greek word “air”, which means breeze. It was first used by Galen. Aura is defined as a ictal phenomenon that in a given patient may precede an observable seizure. If it occurs alone, constitutes a sensory seizure.[21,22]

Ictal semiology; Ictus is defined as a sudden neurological occurrence such as a seizures or stroke. The term semiology means that branch of linguistics concerned with signs and symptoms. Ictal semiology means the symptoms and signs associated with epileptic seizures.[21]

Other common descriptive terminology used in the field of epilepsy, are used in this study as per the definitions of International League Against epilepsy Commission Report.[21]

Classification of seizures and epileptic syndromes;

To understand the epilepsy, we must understand the classification of the epilepsy. It will be the first step for the correct diagnosis, treatment selection and prognostication of the condition. The classification of epileptic seizure is mainly based on clinical observation and opinion of the experts. The current classification of seizures evolved from the early work undertaken in 1960’s, Gastaut H et al., has published this work in 1969 and 1970.[23] It was revised in 1981; this revision classification did not consider brain pathology, age and etiology

instead, it restricted the basis to clinical seizure type in addition to electroencephalography (EEG) data. The International League Against epilepsy (ILAE) 1981 classification of epilepsy was officially updated and published in 1989. The 1981 and 1989 updates are the officially accepted classification system.[24,25]

Differential diagnosis;

Accurate diagnoses of epilepsy in patients with transient neurological events have many implications like psychological issues and therapeutic decisions. Up to 20-30 % cases are misdiagnosed as epileptic seizures (Scheepers et al., 1998; Chadwick et al., 2002). The following are some of the non epileptic events that have to be differentiated from epileptic seizures, which are syncope (orthostatic / arrhythmia, others), migraine (complex), transient ischemic attack, transient global amnesia, sleep disorders, waxing and waning delirium, intermittent movement, panic / anxiety attacks, conversion, psychogenic non-epileptic seizures, hyperventilation syndrome, acute psychosis and malingering.[26,27] The gravity of the problem and the consequences of misdiagnosis can be learned from the case of Dr Andrew Holton.[28]

AIM OF THE STUDY

AIM OF THE STUDY

- To study the clinical profile of epilepsy in patients with onset of epilepsy in the first three years of their life in a tertiary care hospital.

- To study the etiology of epilepsy in patients with onset of epilepsy in the first three years of their life in a tertiary care hospital.

REVIEW
OF
LITERATURE

REVIEW OF LITERATURE

Epileptic seizure is a significant cause for disease burden and disability in the world. As per the estimation of International League Against epilepsy (ILAE) and World Health Organization (WHO) over 50 million people are suffering from epilepsy all over the world. Almost two third of the people with epilepsy are living in developing countries including India and around 80% of them did not receive treatment.[29,30] The population of India is about 1 billion and the expected medically refractory epileptic seizures are about 1 million.[31] Almost 70% of Indian population lives in rural area, where specialist neurological care primarily provided by primary and secondary care physician. Most of the studies have found that the medical treatment gap in epilepsy is around 70 % in India.[29,30,31,32]

The prevalence of epilepsy varies from country to country. It is partly due to different protocols adopted in the diagnosis and classification of people with epilepsy. As per Hauser et al., study the average prevalence rate of epilepsy was 5.2 per 1000 population.[33] The prevalence rate per 1000 population was 2.5, 4.4, and 3.6 for Kashmir, Bangalore and Parsis in Mumbai respectively.[34,35,36] Sritharan and Murthy had estimated that the prevalence rate for urban was 5.1 and for

rural was 5.5. The age adjusted prevalence was 5.3 per 1000 population based on a meta analysis of 20 community based studies in India.[37]

A study done by Thomas SV et al.,[30] at the department of neurology, Sree Chitra Tirunal Institute for Medical Science and Technology, Trivandrum, India showed that the treatment gap was nearly 21%. Since most of their patients were referred from peripheral centers, they had observed that low dose polytherapy was commonly used than high dose monotherapy in patients with poor seizure control. Nearly 25% of referred patients were not on treatment at the time of referral to their institute. As per their observation the treatment gap was associated with traditional medicine use, recent onset of seizures, non disabling nature of patients illness, lack of response to therapy, adverse effects of drugs and higher cost. These observations are much different from the observations of epidemiological studies, where in poor infrastructure, lack of priority, poor availability; high cost and varied perception of disease in different part of the world were the factors.[32] About 57% of the total treatment cost was due to the cost of the drugs. The annual cost of Anti epileptic drug was INR 1898, 8.8% of the per capita Gross National Income (GNI) for monotherapy, but it was 2.5 times INR 4929 for polytherapy. Polytherapy and seizure frequency of 1 or more were affected the quality of life.

K.Radhakrishnann et al., has estimated that the overall age adjusted prevalence rate was 4.7, for males it was 4.9 and for female it was 4.4 per 1000 population in Kerala based on an epidemiological study.[38] In most of western countries including USA and UK the annual incidence rate was around 50 per 100,000 population.[39] The age specific incidence follows a U shaped curve, in which the lowest incidence is in the age group of 30 to 40 years and highest incidence in the elderly people and infants. Almost 40% of epilepsy occurs in children below 15 years, another 40% are in the age group of 15 -64 years of age and around 20% are in elderly people.[39]

Shankar P Saha et al.,[40] have done an incidence study in rural West Bengal, India. As per this study, the age adjusted annual incidence rate is 42.08 per 100,000 population per year. Age specific incidence rate had progressive decline as the age increases except in the age group of 40-49 years where slight increase was found. The authors reviewed that some of the developing countries like Latin America and Africa have high annual incidence rate.

Mani et al., also documented an incidence rate of 49.3 per 100,000 population.[35] The overall incidence and prevalence of epilepsy from various studies are given below (Table-1).[41,42] Epilepsy is slightly more common in males than in females but the difference is not

statistically significant. Most of the studies have found that epilepsy is more common in children living in lower socio economical condition irrespective of their ethnicity (Table-1).[39]

Table-1: Incidence and Prevalence

literature	Incidence (per 100 000 population /year)	Prevalence (per 1000 population)
Western literature[41]	Developed countries :40-70 Developing countries: 100-190	3.3-7.8 -
Asian literature[42]	China : 28.8- 35.0 India : 38-49.3	China: 3.6-4.6 India: 3.8-6.2

V. Udani et al.,[5] have done a study between May 2004 and August 2004. He has studied the etiological aspect of remote symptomatic epilepsy with onset in the first three years of life. During the study period 100 patients were recruited, of which 67 were boys and 37 were girls. The mean age of onset of seizure was 13.9 months in this study. Definitive etiological diagnosis was made in 83 patients. The most common etiology was perinatal encephaloclastic (brain damaging) conditions noted in 50 patients. Of which, neonatal hypoglycemic brain

injury (NHBI) was noted in 23 patients, hypoxic ischemic injury (HIE) was found in 8, periventricular leucomalacia (PVL) in 7 patients, focal cortical infarcts (FI) in 9 patients and multiple etiology in 3 patients. The developmental etiology was found in 28 patients. Of which migration defects in 9 patients, tuberous sclerosis in 9 patients, Aicardi syndrome was in 4, metabolic causes in 3 patients and others in 3 patients. Neonatal hypoglycemic brain injury (NHBI) was the common etiology. 14 out of 23 NHBI patients had documented hypoglycemia in neonatal period; 9 other patients did not have birth records. Microcephaly, visual impairment (cortical) apraxia of hand use and autism were the clinical feature observed in NHBI patients. Spasticity, dystonia were less frequently found in this study. Infantile spasm in 12 patients (52%), partial seizures (22%), generalized seizures (17%) and mixed (9%) were seizure type in these patients. More than 50% of patients had refractory seizures. Risk factors associated with NHBI were LSCS delivery, birth weight less than 2.5 kg and poor feeding in neonatal age. They have observed that even babies with appropriate for gestational age (AGA) had NHBI. Late new born feeding might be the predisposing factors for NHBI. The causes of infantile remote symptomatic epilepsy, in developing countries, is related to perinatal brain injuries whereas in developed nations these are mainly due to

developmental malformations like cortical dysplasia, tuberous sclerosis etc.

Thomas Varghese Attumalil et al.,[43] have done a study at government Medical College, Trivandrum, Kerala. They have examined 4 broad categories of risk factors for epilepsy (familial factors, maternal factors, perinatal factors and postnatal factors). Newborn distress, developmental delay, head trauma and family history were the risk factors significantly associated with epilepsy, which account for 40% of the risk of epilepsy in children. In this study the prevalence of consanguinity in the epilepsy patients was 13.4% as against the national prevalence of 15.9% to 32.9% (mean 22.2%). Maternal factors like consanguineous marriage, age of the mother at delivery, recurrent abortions, antenatal infections, gestational hypertension, gestational diabetes were not associated with development of epilepsy. Newborn distress was associated with early onset of epilepsy.

Huseyin Per et al.,[44] have studied the neurological sequelae associated with newborn hypoglycemia. Grade 1 hypoxia, prematurity, hyperbilirubinemia, polycythemia, sepsis, exchange transfusion, preeclampsia, eclampsia, intrauterine growth retardation, diabetic mothers, oligohydramnios and congenital heart disease were associated with newborn hypoglycemia. Endocrine disorder like cortisol

deficiency, hypothyroidism, hyperinsulinism, hyperammonia also accompanied the hypoglycemia. Epilepsy, mental retardation, microcephaly, autistic behavior and attention deficit hyperkinetic disorders were the observed neurological sequelae. MRI imaging of these patients showed evidence of brain injuries in parieto-occipital region, occipital region, parietal region, cystic encephalomalacia, cortical atrophy, fronto-temporal region and periventricular leucomalacia in descending order of frequency. Some patients had normal imaging, in which epilepsy was the only neurological complication found. They concluded that hypoglycemia often coexists with birth asphyxia, which may lead to more severe neurological damage. High risk newborns have to be closely monitored during the first 3 days of life to avoid these complications.

Teodoro Dura-Trave et al.,[45] have done a study in Navarre, Spain among children younger than 15 years of age. They have observed a high annual incidence rate of epilepsy during the first year of life (95.3 per 100,000 population), then it decreases till adolescence. In infants (1-12 months) group, symptomatic epilepsy was noted in 63.6%, cryptogenic in 43.9% and idiopathic epilepsy in 9.1% of patients. In early childhood (1-6 years) group, the symptomatic seizures were present in 25.8%, cryptogenic were present in 43.9% and idiopathic

accounts for 30.5% of etiology. In this cohort, the family history of epilepsy was 24.1% and the personal and family history of febrile seizure in 13.6%. The authors have found that the focal epilepsies were present in 55% of patients, generalized epilepsies in 42.9% and undetermined localization in 2.1%. In different study by same authors have observed that the complex seizures were 28.7% and complex with secondary generalization were 16.35 in focal epilepsies category. Typical absence seizures were 14.3% and tonic-clonic seizures were 10.2% in generalized epilepsies group.[46]

Javad Akhondianet al.,[47] have done a case-control study in children below 15 years of age. In 64.7% of children with intractable seizures (cases), the age of onset seizures was under 1 year. Positive family history for epilepsy was 13.7%, 12.5% in intractable (cases) and well controlled seizures (control) group respectively. In this study 19.6% of case and 22.5% of controls had focal seizures, 66.7% of cases and 75% of controls had generalized seizures at the onset and 13.7% of cases and 2.5% of controls had myoclonic seizures at the onset. Neurological deficit was present in 80.4% of cases and 8.8% of controls ($p<0.001$). Another observation in this study was that 66.7% of cases and 22.5% of controls had daily seizures, 9.8% of cases and 8.8% of controls had more than one episode per week, 19.4% of cases and 24.5%

of controls had 1-4 episodes of seizures per month, 3.9% of cases and 41.3% of controls had less than 1 attack per month ($p<0.001$). Neonatal seizures were found in 17.6% of cases and 5% of controls ($p<0.018$). History of status epilepticus was present in 11.8% of cases and 11.3% of controls ($p =0.018$). The mean age of presentation was 19.6 months, 46.5 months in cases and controls respectively ($p=0.002$). In males, the age of onset was 16.7 months in cases and 48.6 months in control group ($p =0.003$) and in females this was 27.8 months in cases and 44.2 months in controls ($p = 0.216$). It was also found that 96.1% of cases and 83.85% of controls had abnormality in their 1st electroencephalogram (EEG) ($p<0.031$). Computerized tomography (CT) was abnormal in 52.9% in cases and 13.5% in control group ($p = 0.002$).The other observations were male sex, onset of seizure under the age of 1 year, myoclonic seizures, daily seizures, history of newborn seizures, presence of neurologic deficit and abnormal imaging are associated with increased risk of intractable seizures.

Christine M. Freitag et al.,[48] have done a prospective (population based) study in German children aged between 1month to less than 15 years. The annual incidence rate was high in younger children (1 month to 12 months) and 22.2% of children had first degree relatives who had epileptic seizures in them. Idiopathic epilepsy was

present in 47.2% of children, symptomatic or cryptogenic was in 50%. The idiopathic etiology was more commonly associated with generalized epilepsy than focal epilepsy. In this study 11% of children had central nervous system malformation, 5.6% had perinatal complications, 13.8% had severe mental retardation (1 child with angelman syndrome, 2 with dimorphic syndromes) and 5.6% of children had developmental delay. Carbamazepine was the initial drug used in 53.1% of children, sodium valproate was used in 40.6% of cases and 4 children didn't receive treatment. Focal epilepsies were diagnosed in 58% of children, generalized epilepsies in 39% of case and undetermined seizure type in 3% of cases.

Sanjeev V Thomas,[49] in his review article "prevention of epilepsy and obstetric care, has reviewed studies relating to perinatal factors and risk of epilepsy (Table- 2). He also concluded that nearly 10% of incident epilepsies are potentially preventable and in developing countries, 60% of deliveries are not attended by trained persons (WHO and ILAE estimation).

Table-2(a): Review of study by Sanjeev V Thomas

Study	Conclusions
Follow up studies: 1)National Collaborative perinatal project (NCPP)(Nelson et al.,)[50,51]	-Labor and delivery factors contribute very little to childhood seizures. Brain maldevelopment contributes to seizures.
2)Tsuboi and Okada[52]	-No significant association.
3)Rantakallio P et al., Finland[53]	Prenatal factors had highest relative risk for all subtype of epilepsies. Perinatal and postnatal factors had lower relative risk.
4)British national child development study(Kurtz et al.,)[54]	No specific obstetric risk factors.
Case control study: 1)Rocca et al., Rochester Minnesota[55,56]	None of the perinatal factors were significantly associated with CPS or GTCS.
2)Monetti VC, Casetta et al.,[57,58]	Family history of epilepsy, maternal age >35 years, birth order >3 and continuous physical activity during pregnancy had association but in their subsequent study this association was not present.
Sidenvall R et al., Sweden[59]	Vaginal bleeding, gestational age and cesarean section had significant association for epilepsies. Smoking during pregnancy was a risk factor for unprovoked seizures.
Other study: 1)Al-Rajeh S et al., Saudi Arabia[60]	Perinatal encephalopathy was responsible for 40% of the epilepsies in children under 5 years.
2)Haekett R J et al., Kerala, India[61]	Perinatal complications, low BMI, recent physical symptoms had association with active epilepsies.
Kerla et al., India[62]	66% of infantile spasm had pre or perinatal etiological factor.
Massuo A et al., Japan[63]	83% had symptomatic infantile spasm, in which prenatal factor (most common), low birth weight (LBW), perinatal and postnatal factor were noted as etiological factor.
Studies from Bengal[64] and Chandigarh[65]	No association found.

H. M. Hamer et al.,[66] have done a study among children younger than 3 years who had epileptic seizures during prolonged video- EEG monitoring at the Cleveland clinic foundation, Ohio, USA. Based on video- EEG they have described a semiologic classification of seizures: 1) Tonic seizures, 2) Myoclonic seizures, 3) Clonic seizures, 4) Atonic seizures, 5) Versive seizures, 6) Epileptic spasms, 7) Hypomotor seizures, 8) Automotor seizures, 9) Unclassified motor seizures. In this study EEG seizures were classified for clinical purpose as 1) focal, 2) lateralized, 3) generalized or nonlocalized. On the basis of clinical and laboratory information epilepsies were classified as 1) Focal epilepsy, 2) Multifocal epilepsy, 3) Generalized epilepsy and they were further characterized as idiopathic epilepsies, cryptogenic epilepsies and symptomatic epilepsies. In this study, symptomatic epilepsies were defined as epilepsies involving an underlying brain lesion, which was visible in neuroimaging or other central nervous system pathology that precipitated the seizures. Cryptogenic seizures: probably symptomatic seizures, but the specific cause not identified. Idiopathic epilepsies: they were associated with normal neurologic and neuroimaging findings and probable hereditary components. Motor seizures were the common type, which accounted for 79% of seizures. Hypomotor seizures accounted for 20%, unclassified were 8% and Automotor seizures were 1%. Tonic,

clonic seizures, epileptic spasm and hypomotor seizures together accounted for 81% of total seizure. Aura and typical GTCS evaluation were not seen in this study. Symptomatic epilepsies were seen in 70% of patients, cryptogenic in 29% and idiopathic epilepsies in 1%.

MATERIALS AND METHODS

MATERIALS AND METHODS

Study Design : Descriptive study.

Study population : People with epilepsy attending outpatient clinic (OPD) in Department of Neurology & Paediatric Neurology at Government Stanley Medical College, Chennai.

Study period : February 2012 to January 2013.

Place of Study : Department of Neurology and
Department of Paediatric Neurology,
Govt. Stanley Medical College, Chennai,
Tamil nadu.

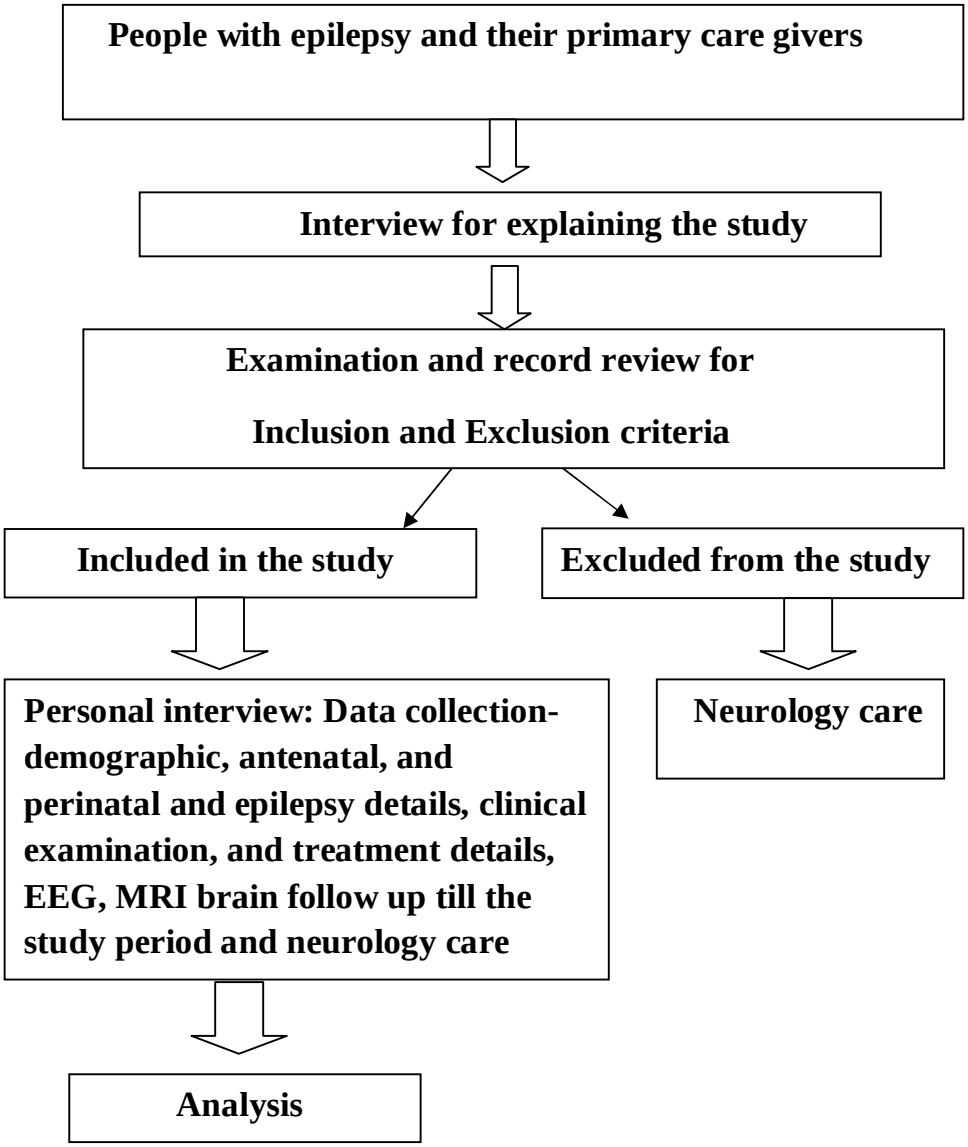
Inclusion Criteria:

All epileptic patients with onset of seizure within the first three years of their life and continue to have seizures (2 or more seizures), irrespective of their present age.

Exclusion Criteria:

- Febrile seizure patients
- Epilepsy following febrile seizures
- Patients with seizure onset above the age of three years
- Uncertain about the age of onset of seizures
- Patient without MRI brain imaging
- Patient unwilling to participate in this study were excluded.

Methodology:



This study is done at the outpatient (OPD) Department of Neurology and outpatient (OPD) Department of Paediatric Neurology at Government Stanley Medical College, Chennai, Tamil nadu, India.

All epilepsy patients (both newly registered as well as patients already on follow up) and their primary care givers attending epilepsy clinic were interviewed for inclusion and exclusion criteria. Patients excluded from the study were explained the reasons and were sent for regular care. Patients fulfilling the inclusion criteria were included in the study after getting informed consent from the patient or their parents / guardian. Patients and their primary care givers were explained about the nature of the study and the need of regular follow up with investigator. They were encouraged to ask all their doubts and report all their health problems including recurrence of seizures, drug side effects and other medical help. They were treated as per the institutional policy.

Name, age, sex, education, scholastic performance, socio-economical status, consanguinity of parental marriage, family history of seizures and febrile seizures were obtained from patient and parents or from primary care givers. Detailed antenatal, natal, and neonatal history was obtained from parents or primary care givers. Antenatal history includes antenatal registration, antenatal care, antenatal events like fever, bleeding, previous abortions, medical illness, treatment history, drug intake by mother. Perinatal history includes home/hospital delivery; persons conducted the delivery and nature of delivery like normal vaginal delivery /LSCS/forceps delivery. Neonatal history

includes gestational age (preterm, term, post term), birth weight, newborn admission, newborn seizure and altered sensorium, hypoglycaemia, the day it occurred, feeding difficulties, details of investigations, treatment etc.,. The age of onset of seizure details and developmental milestones were obtained. The habitual seizure semiology, seizure frequency and presence of status epilepticus were documented. Other relevant data including treatment, drugs, duration of treatment, and its response and adverse effect were collected. Detailed general examination, neurological examination was done. All patients included in this study underwent 1.5 tesla MRI Brain at our institution. In our study, the etiological diagnosis was made based on MRI brain. The imaging findings were divided into 1) Normal 2) Perinatal encephaloclastic (PEC) conditions, which include hypoxic ischemic encephalopathy (HIE) changes, neonatal hypoglycemic injuries (NHBI), periventricular leucomalacia (PVL), and focal infarcts (FI) 3) Other etiology (like mesial temporal lobe sclerosis, tuberous sclerosis, focal cortical dysplasia, heterotopia etc.,). Two Radiologists reviewed the MRI brain and suggested a probable etiological diagnosis. Doubtful cases were discussed and final diagnosis was given on consensus basis. Interictal surface Electroencephalography (EEG) was done for all patients included in this study by using 10-20 system. The EEG was

reported by an Epileptologist in our institution. Other relevant investigations were done as per the clinical need and treating neurologist advice. All patients received appropriate treatment as per institutional policy. All these patients were followed up during study period and medical events were documented.

Statistical method;

All these data were coded and entered into excel sheet and detailed analysis of the data was done by using SPSS-PC windows version 16.0. The Pearson Chi-Square test and student independent‘t’ test were used wherever applicable and P-value less than 0.05 was taken as significant.

OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

Totally 115 patients were included in our study.

Age:

The youngest patient was one year, the oldest patient was 36 years of age and the mean age was 11.4 ± 7.58 years.11 (9.56%) patients were between 1 to 3 years, 23 (20%) patients were between 3 to 6 years, 34 (29.56%) patients were between 6 to 12 years, and 35 (30.44.56%) patients were between 12 to 21 years (Table-3).

Table-3: Age distribution of the study population

Age	No. of patients (%)
1 - 3 Years	11 (9.56%)
3 - 6 Years	23 (20%)
6 - 12 Years	34 (29.56%)
12 - 21 Years	35 (30.44%)
More than 21 years	12 (10.44%)

Sex:

In this study, 68 (59.10%) patients were males and 47 (40.90%) patients were females (Table-4).

CHART-1: DEMOGRAPHY AND FAMILIAL FACTORS

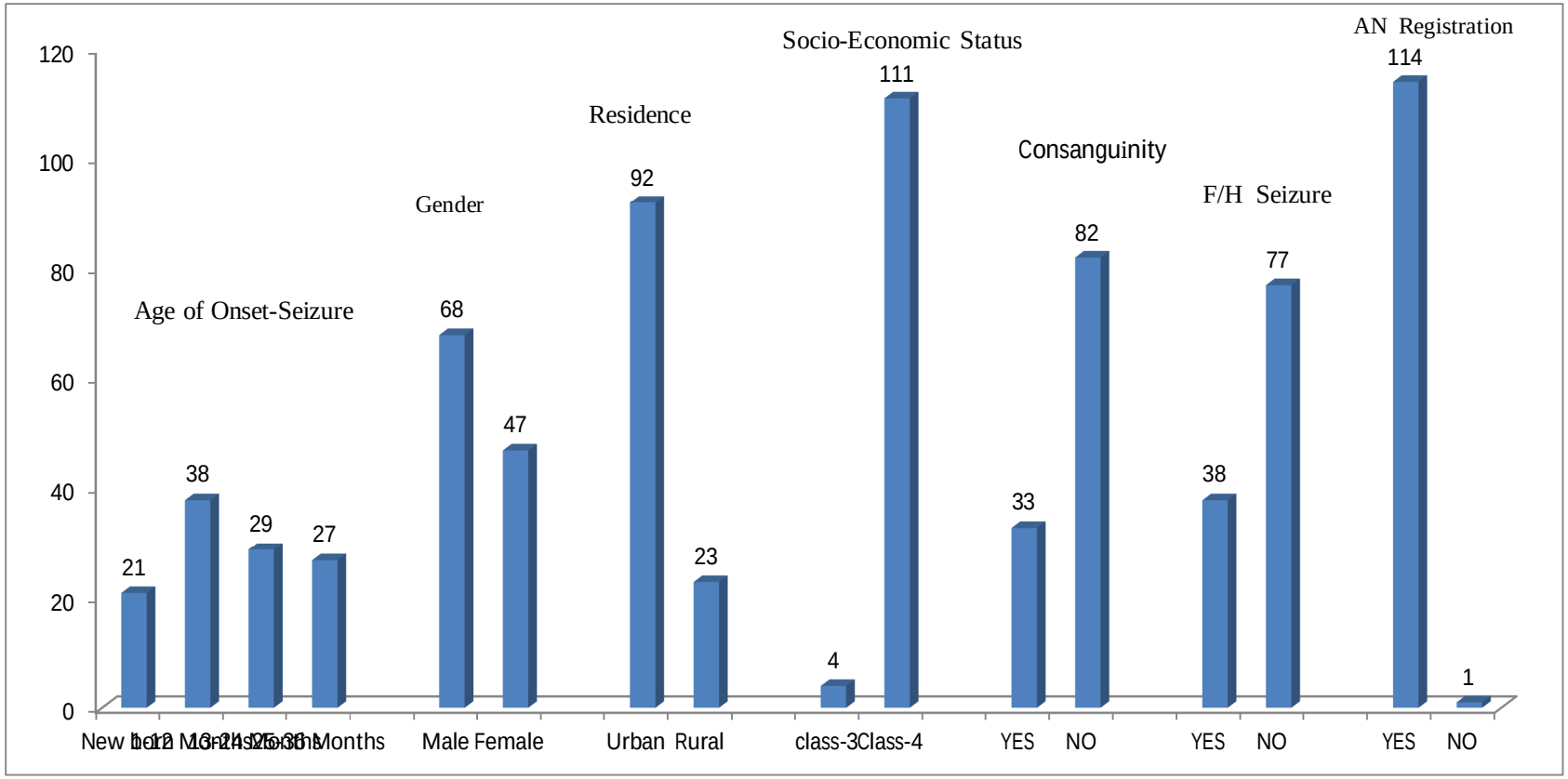


Table-4: Sex of the study population.

Sex	Male	Female
No. of patients (%)	68 (59.10%)	47 (40.90%)

Age of onset seizures:

21 (18.26%) patients had seizure onset in the newborn period, 38 (33%) patients had seizure onset between 1 to12 months of age, 29 (25.21%) had onset of seizures between13 to 24 months of age and 27 (23.5%) had between 25 to 36 months of age (Table-5). The mean age of onset of epilepsy was 14.8 ± 11.2 months.

Table-5: Age of onset seizure in the study population

Seizures onset age	No. of patients (%)
Newborn period	21 (18.26%)
1 – 12 months of age	38 (33%)
13 – 24 months of age	29 (25.21%)
25 – 36 months of age	27 (23.53%)
The mean age of onset	14.8 ± 11.2 months

Residence, Socio Economic Status and Marital status:

92 (80%) patients were from urban area and 23 (20%) were from rural area. As per updated kuppusamy's scale (2007), 111 (96.50%) patients belong to class-4 socio economical status and 4 (3.50%) patients belong to class-3 category. In our study 12 patients were above the age of 21 years and six of them were married (Chart-1).

Literacy:

Table-6: Literacy status

Literacy	No. of patients (%)
Illiterate	12 (10.43%)
1^{st} to 5^{th} std.	43 (37.39%)
6^{th} to 12^{th} std.	31 (26.96%)
Completed 12^{th} std	3 (2.61%)
Yet to join school	26 (22.61%)

Std-standard (Class)

Among our study population, 12 (10.43%) patients did not know to read and write, 43 (37.38%) patients were attending primary school or studying in primary school at the time of stopping from school, 31 (26.96%) patients were attending 6^{th} -12^{th} class or studying in 6^{th} -12^{th} classes at the time of stopping from school, 26 (22.61%) patient yet to

join primary school and one patient was studying in college. On further analysis, we found 18 patient's age were 18 years and above and only 3 (2.64%) of this patients have completed higher secondary school and 1 of the patient was studying in college (Table-6).

Consanguinity:

33 (28.7%) patients were born to consanguineous parents (table-7).

Table-7: family history seizures, Consanguinity and Antenatal registration

Maternal factor	Yes	No
Consanguinity	33 (28.7%)	82 (71.3%)
Family history of seizures	38 (33%)	77 (67%)
Antenatal registration	114 (99.1%)	1 (0.9%)

Family history of seizures:

38 (33%) patients had family history of seizures and 77 (67%) patients did not have family history of seizures (Table-7). Only 2 (1.7%) patients had family history of febrile seizures (mother-1 and female sibling-1).

Antenatal registration:

114 (99.1%) patient's mother were registered during antenatal period (114 patients were born out of registered pregnancy who had regular antenatal checkups) and 1 (0.9%) patient's mother did not have antenatal registration (Table-7).

Maternal high risk factors:

2 (1.7%) mothers had eclampsia, 3 (2.6%) mothers had diabetes / gestational diabetes, 1 (0.9%) mother had antepartum bleeding and 18 (15.7%) mothers had recurrent abortions before the delivery of the index cases (study population).

Place of delivery:

Table-8(a): Place of delivery

Place of delivery	Institutional delivery	Home delivery
No. of patients (%)	107 (93%)	8 (7%)

CHART-2: MATERNAL AND PERINATAL FACTORS

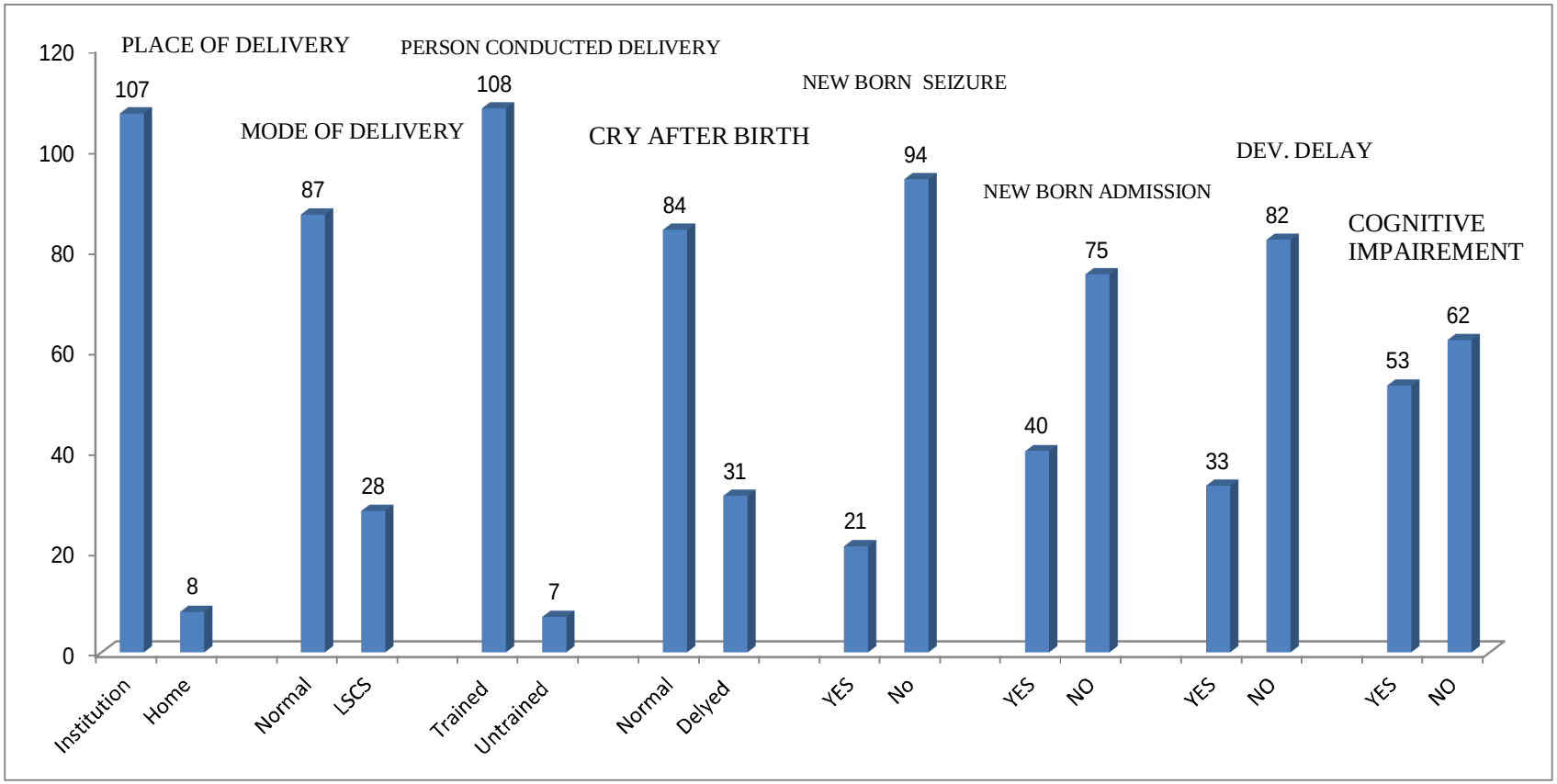


Table-8(b): Institutional delivery

Institution	No. of patients 107 (93%)
Medical Colleges (MC)	53 (46.1%)
Government Hospitals (GH)	18 (15.7%)
Primary Health Centers (PHC)	15 (13%)
Subcenters (SC)	5 (4.3%)
Private Hospitals (PVT)	16 (13.9%)

107(93%) patient's mothers had institutional delivery and 8(7%) had home delivery. Among the institutional deliveries, 53(46.1%) deliveries were at medical colleges (MC), 18(15.7%) were at government hospitals (GH) at district and taluk level, 15(13%) were at primary health centers (PHC), 5(4.3%) were at subcenters (SC) and 16(13.9%) were at private hospitals (Table 8 (a, b).

Mode of delivery:

87 (75.7%) patients were born of vaginal delivery and 28 (24.3%) patients delivered by lower segment caesarian section (LSCS) (table-9). The indications for caesarian section were repeat LSCS in 15 (13%) deliveries, fetal distress in 6(5.2%) deliveries, obstructed labour in 1(0.9%) and eclampsia in 1(0.9%).

Table-9: Mode of delivery

Mode of delivery	Vaginal delivery	LSCS delivery
No. of patients (%)	87 (75.7%)	28 (24.3%)

Person conducted delivery:

The deliveries were conducted by trained persons (Doctors, Nurses, Village Health Nurses) in108 (93.9%) and by untrained persons in 7 (6.1%)(Table-10).

Table-10: Person conducted delivery

Person conducted delivery	Trained person	Untrained person
No. of patients (%)	108 (93.9%)	7 (6.1%).

Birth weight and gestational age:

The birth weight was less than 2.499 kg in 24 (20.9%) patients, 83 (72.2%) patients had birth weight between 2.5 to 3.499 kg, and 7 (6.1%) patients had birth weight between 3.5 to 4 kg and 1 (0.9%) patient had birth weight above 4 kg. Of this, 110 (95.7%) patients were term and 5 (4.3%) patients were preterm (Table-11).

Table-11: Birth weight

Birth Weight	< 2.499 Kg	2.5-3.499 kg	3.5 – 4 kg	> 4Kg
Patient n (%)	24 (20.9%)	83 (72.2%)	7 (6.1%)	1 (0.9%)

Cry after birth:

84 (73%) patients had normal cry after birth and 31 (27%) patients had delayed cry after birth (table-12).

Table-12: Delayed cry after birth in study population

Cry after birth	Cried after birth	Delayed cry after birth
No. of patients (%)	84 (73%)	31 (27%)

Newborn feeding:

Newborn feeding was started within 3 hours of birth in 76 (66.1%), feeding was started between 3 to 6 hours of birth in 8 (7%) and later in 31 (27%). 72 (62.61%) patients were given breast feeding as the first feed, 14 (12.17) patients were given pre-lacteal /artificial feeds and 29 (25.18%) patients were on intravenous fluid therapy (Table-26).

Newborn seizure:

21 (18.3%) patients had history of newborn seizures and 94 (81.7%) patients did not have history of newborn seizures. The onset of newborn seizures was within 24 hours of birth in 9 (7.8%) patients, between 24 to 72 hours of birth in 5 (4.3%) patients, between 4 to 7 days of birth in 4 (3.5%) patients and between 8 to 30 days of birth in 3 (2.6%) patients (Table-13).

Newborn admission:

Table 13: Patient characters

Patient variable	Present in n (%)
Newborn seizures	21 (18.3%)
Newborn admission	40 (34.8%)
Developmental delay	33 (28.7%)
Cognitive impairment	53 (46.1%)
Psychosis	10 (8.7%)
Neurological deficits	10 (8.7%)
Facial dysmorphism Microcephaly Neurocutaneous markers	2 (1.7%) 5 (4.3%) 2 (1.7%)

In our study, 40 (34.8%) patients had history of newborn admission and the duration varied between few hours to 7 days (table-13).

Developmental history, Cognitive impairment and Psychosis:

82 (71.3) patients had normal developmental milestones, 33 (28.7%) patients had delayed developmental milestones. 2 patients had mild language delay alone. 53 (46.1%) patients had cognitive impairment and 10 (8.7%) patients had psychosis (table-13) (Chart-2).

Neurological deficits:

2 (1.7%) patients had facial dysmorphism and 2 other patients had neurocutaneous markers. 10 (8.7%) patients had focal neurological deficits with spasticity and 5 patients had microcephaly. One patient had features of tuberous sclerosis. One patient's mother had Gilbert syndrome and one patient's father had thalassemia (table-13).

Habitual seizures characters:

In our study 12 (10.2%) patients had aura (visual-4, auditory-1, sensory-5, and smell-2), 22 (19.14%) patients had history of status epilepticus (SE) in the past and 13 (11.3%) patients had clustering episode of seizures. 7 (6.1%) patients had nocturnal seizures, 73

(63.5%) patients had day time seizures and 34 (29.6%) patients had seizures during both day and night time (Table-14) (Chart-3).

Table14: Seizure character

Seizure character	Present
Aura	12 (10.2%)
Status epilepticus (SE)	22 (19.14%)
Clusters	13 (11.3%)
Nocturnal seizures	7 (6.1%)

Epilepsy type:

2 (1.7%) patients had simple partial seizures (SPS), 57 (49.6%) patients had complex partial seizures (CPS), 34 (29.5%) patients had complex partial seizures with secondary generalization, 21 (18.3%) patients had generalized seizures and 1 patient had gelastic seizure (table-15). Of the above 93 (80.9%) focal epilepsies, 30 (26.1%) patients had complex partial seizures of temporal origin (CPS-T) and 63 (54.8%) patients had complex partial seizures of extra temporal origin (CPS-ET) (Table-15).

CHART-3: CLINICAL AND EPILEPSY FACTORS

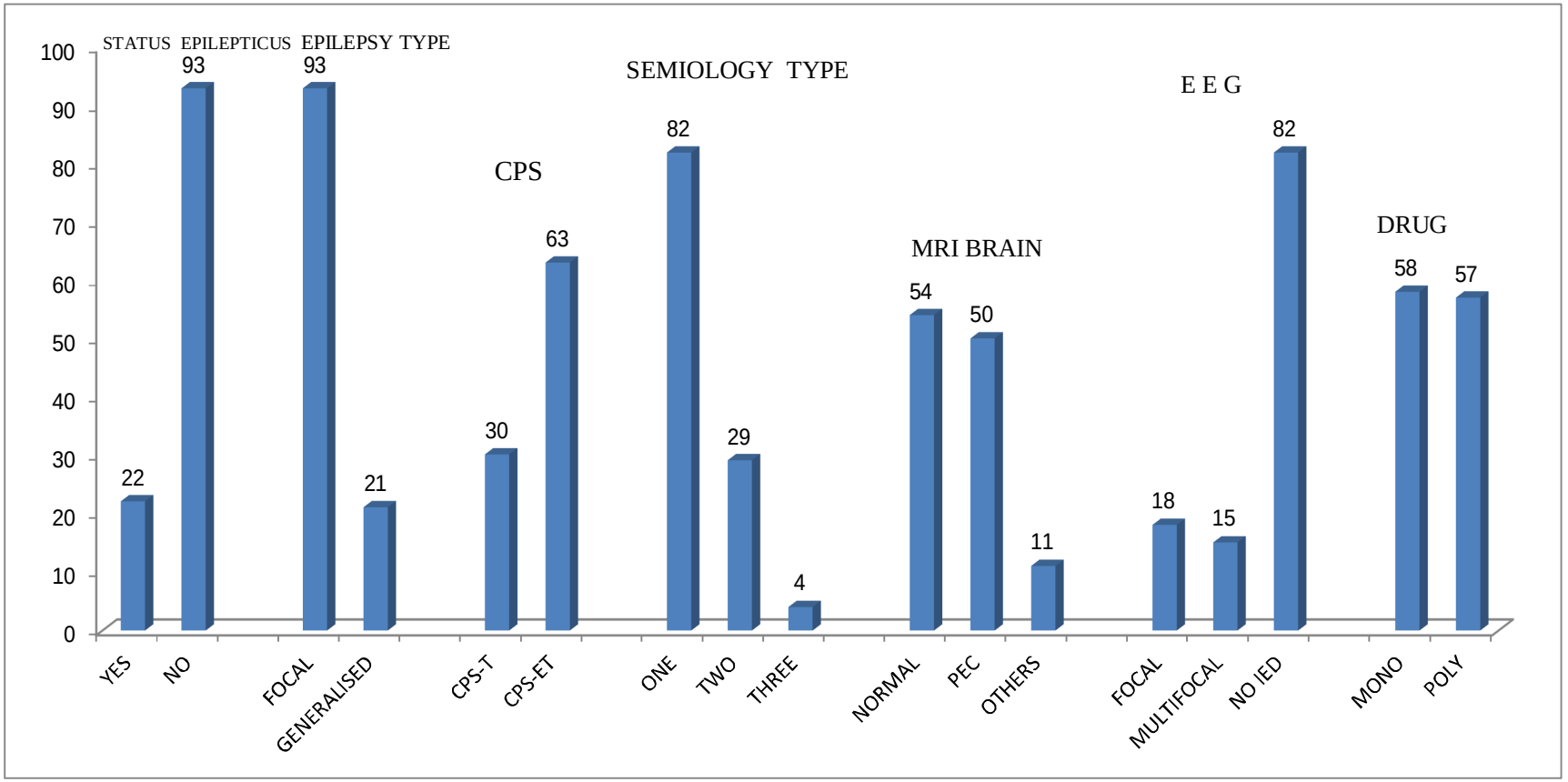


Table-15: Epilepsy type

Epilepsy type	No. of patients (%)
Simple partial seizures	2 (1.7%)
Complex partial seizures	57 (49.6%)
Complex partial seizures with secondary generalization	34 (29.5%)
Generalized seizures	21 (18.3%)
Gelastic seizure	1 (0.9%)
Subtype; CPS-Temporal origin (T) CPS-Extra temporal (ET)	 30 (26.1%) 63 (54.8%)

Frequency of Seizures:

15 (13%) patients had at least one episode of seizure every week, 20 (17.4%) patients had at least one episode of seizure every month, 17 (14.8%) patients had at least one episode of seizure every 3 months, 8 (7%) patients had at least one episode of seizure every 6 months, 12 (10.4%) patients had at least one episode of seizure every year, 43 (37.4%) patients had occasional episode of seizures(table-16). While comparing this seizure frequency with the seizure frequency during treatment, there is a 2.6% reduction in weekly seizure frequency after

treatment, 7.8% reduction in monthly seizure frequency and 27 (23.5%) patients did not have seizures during last one year. The seizure frequency was increased by 4.3% at 3months and 8.3% at 6 months respectively (Table-16).

Table -16: Seizure frequency

Seizure frequency	Before treatment	On treatment
At least once per week	15 (13%)	12 (10.4%)
At least once per month	20 (17.4%)	11 (9.6%)
At least once in 3 months	17 (14.8%)	22 (19.1%)
At least once in 6 months	8 (7%)	18 (15.7%)
At least once in year	43 (37.4%)	25 (27.7%)
Occasionally/ no seizures	Occasional-43 (37.4%)	Nil sz 27 (23.5%)

Nil Sz- no seizures for past one year

Seizure free period:

Seizure free period between the 1st and 2nd (habitual) seizures was less than a year in 20 (17.4%) patients, 1 to 5 years in 32 (27.8%)

patients, 5 to 10 years in 7 (6.1%) patients, more than 10 years in 10 (8.7%) patients and 46 (40%) patients did not have seizure free period.

Semiological seizures classification (SSC):

We applied Semiological seizures classification defined by H. M. Hamer et al (H. M. Hamer et al., Epilepsia-1999) to the ictal semiology elaborated by our patients and their primary care givers. As per this, 48 (41.7%) patients had clonic seizures; tonic seizures were present in 23 (20%) patients, 13 (11.3%) patients had epileptic spasms, hypomotor seizures in 11 (9.6%) patients, 6 (5.2%) patients had atonic seizures, versive seizures were present in 4 (3.5%) patients, Automotor seizures were present in 4 (3.5%) patients, unclassified motor seizures in 5 (4.3%) patients and one patient had myoclonic seizure (Table-15).

Motor seizures (tonic, clonic, myoclonic, epileptic spasms, atonic, versive and unclassified motor seizures) were observed in 100 (87%) patients. Hypomotor seizures in 11 (9.6%) and Automotor seizures were present in 4 (3.5%) patients (Table-17).

Table-17: Semiological seizure type

Semiological seizure type	No. of patients (%)
Tonic seizure	23 (20%)
Myoclonic seizure	1 (0.9)
Clonic seizure	48 (41.7%)
Atonic seizure	6 (5.2%)
Versive seizure	4 (3.5%)
Epileptic spasm	13 (11.3%)
Hypomotor seizure	11 (9.6%)
Automotor seizure	4 (3.5%)
Unclassified motor seizure	5 (4.3%)

Number of seizure semiology in each patient:

Table-18: Number of seizure semiology in each patient

No. of seizure type	One	Two	Three
No. of patients (%)	82 (71.3%)	29 (25.2%)	4 (3.5%)

Single seizure semiology type were present in 82 (71.3%) patients, two seizure semiology types were observed in 29 (25.2%)

PERINATAL ENCEPHALOCLASTIC CONDITIONS (PEC)

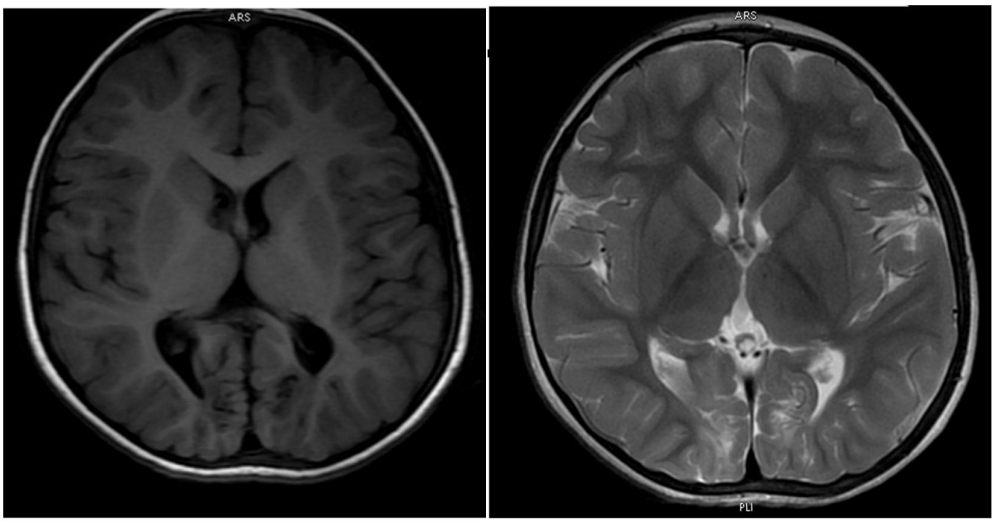


Fig 1 : MRI Brain : T1 and T2WI axial sections shows perinatal hypoglycaemic and hypoxic occipital injuries.

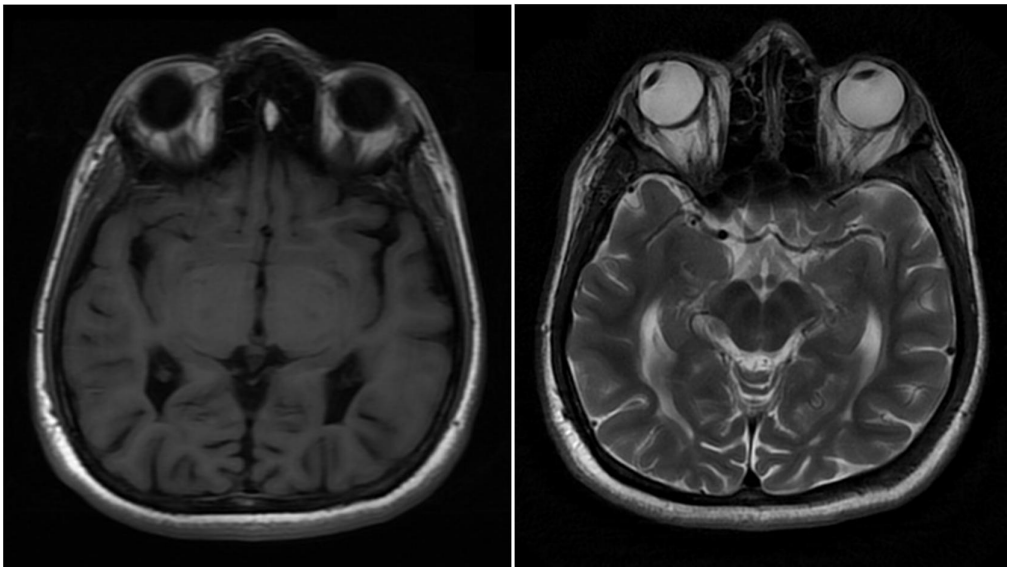


Fig 2: MRI Brain T1 & T2 Axial showing Periventricular Leucomalacia (Undulating ventricular margin)

patients, three seizure semiology type were noted in 4 (3.5%) patients (Table-18).

MRI Brain (Magnetic Resonance Imaging):

In our study, the etiological diagnosis was made based on MRI brain. The imaging findings were divided into 1) Normal 2) Perinatal encephaloclastic (PEC) conditions, which include hypoxic ischemic encephalopathy (HIE) changes, neonatal hypoglycemic injuries (NHBI), periventricular leucomalacia (PVL), and focal cortical infarcts (FI) 3) Other etiologies like mesial temporal sclerosis, tuberous sclerosis, focal cortical dysplasia, heterotopia etc.

In our study, 54 (46.96%) patients had normal MRI brain (non lesional) and the imaging was abnormal in 61 patients. 50 (43.5%) of this patients had perinatal hypoxic-hypoglycemic injuries to the brain (PEC), mesial temporal sclerosis were found in 4 (3.5%) patients, heterotopias were noted in 2 (1.7%), focal cortical dysplasia in 1 (0.9%), hypothalamic hamartoma in 1 patient, cortical tuber in 1 patient, metachromatic leukodystrophy in 1 patient, Fahr's disease in 1 patient (table-19) (Figures 1-6).

PERINATAL ENCEPHALOCLASTIC CONDITIONS (PEC)

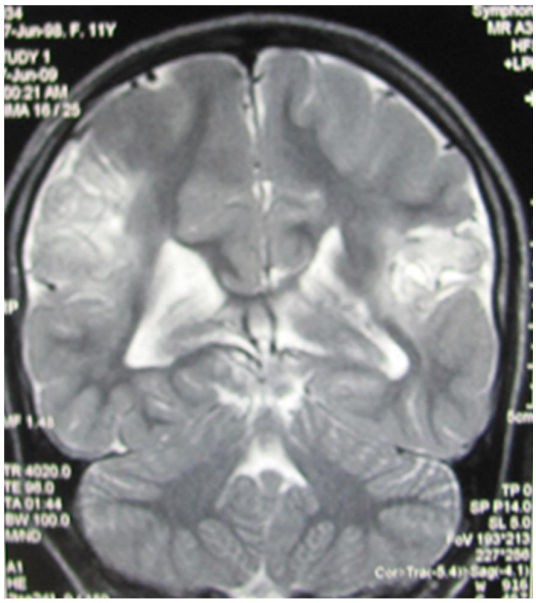


Fig 3: MRI Brain T2 Coronal showing bilateral Perisylvian HIE

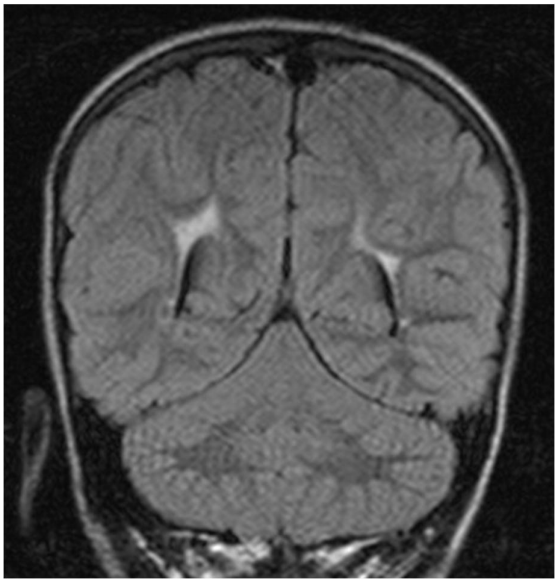


Fig 4: MRI Brain FLAIR Coronal showing bilateral Parieto-occipital HIE

Table-19: MRI Brain

MRI -Brain	Patient n (%)
Normal	54 (46.96%)
Perinatal encephaloclastic (PEC) conditions	50 (43.5%)
Mesial temporal sclerosis	4 (3.5%)
Heterotopia	2 (1.7%)
Focal cortical dysplasia	1 (0.9)
Tuberous sclerosis	1 (0.9%)
Fahr's disease	1 (0.9%)
Metachromatic leukodystrophy	1 (0.9%)
Hypothalamic hamartoma	1 (0.9%)

Posterior head (occipital, parieto-occipital, parieto-occipital with perirolandic) region were predominantly affected in hypoxic-hypoglycaemic injuries of brain (Table-20).

DEVELOPMENTAL DISORDERS

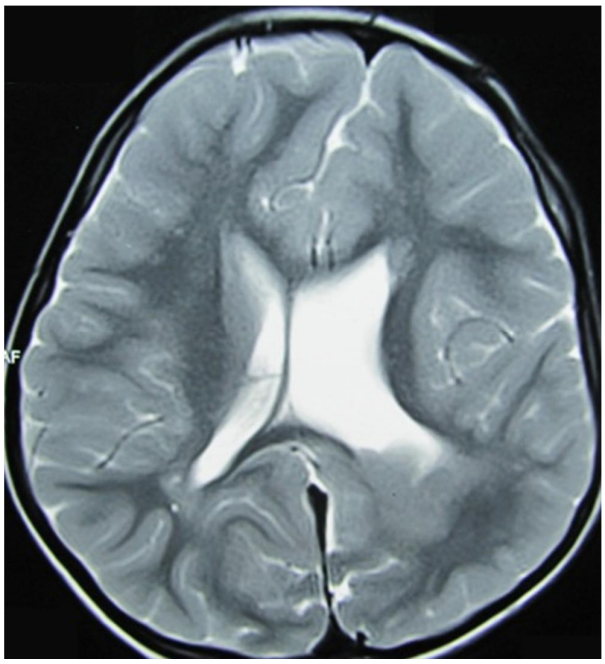


Fig 5: MRI Brain T2 Axial showing Left Parieto-occipital subependymal Heterotopia with Pachygyria

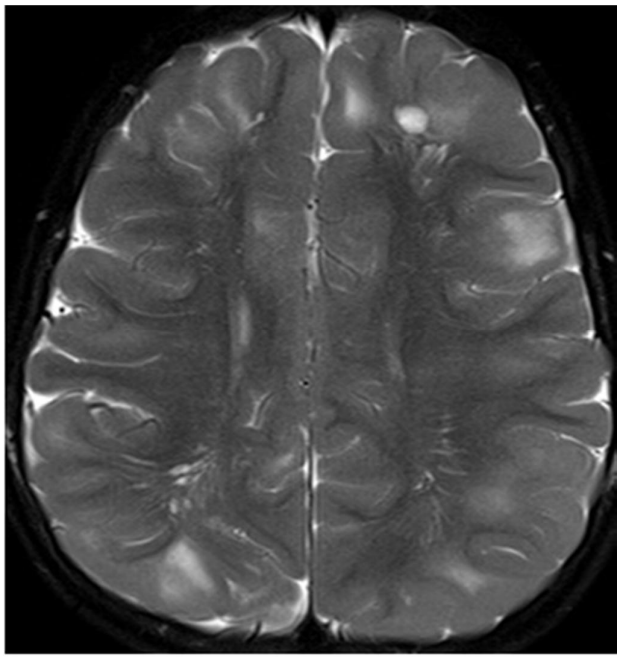


Fig 6: MRI Brain T2 Axial showing cortical tubers

Table-20: Perinatal encephaloclastic (HIE/NHBI) lesions

Site of lesion	Number 50 (100 %)
Occipital region	4 (8%)
Parieto-occipital region	16 (32%)
Perirolandic region	4 (8%)
Perirolandic and parieto-occipital	2 (4%)
All region	15 (30%)
Periventricular	3 (6%)
Frontal, fronto-parietal, parietal, cerebellum	6 (12%)

Etiological classification of epilepsy:

In our study, symptomatic epilepsy was observed in 61 (53.04%) patients, the remaining 54 (46.96%) were non lesional epilepsies.

Electroencephalography (EEG):

18 (15.65%) patients had focal interictal epileptiform discharges (IED), 15 (13.04%) patients had multifocal interictal epileptiform

discharges (IED) and 82 (71.31%) patients did not have interictal epileptiform discharges (IED).

Treatment:

58 (50.4%) patients were on single antiepileptic drug (AED) (monotherapy) and 57 (49.6%) patients were receiving 2 or more antiepileptic drugs (polytherapy). 40 (34.8%) patients were on 2 drugs, 12 (10.4%) patients were taking 3 drugs, 4 (3.5%) patients were on 4 drugs and 1 (0.9%) patient was on 5 drugs(Table-21).

Table-21: Drug therapy

Drug therapy	Monotherapy	Polytherapy
No. of patients (%)	58 (50.4%)	57 (49.6%)

15 (13%) patients were on Phenytoin, 49 (42.6%) patients were on Carbamazepine, 65 (57.4%) patients were on Sodium valproate and 29 (25.2%) patients were taking Phenobarbitone either alone or in combination with other drugs. Other add on drugs were Clonazepam in 7 (6.1%) patients, Clobazam in 7 (6.1%), Levetiracetam in 5 (4.37%) patients, Diazepam in 3(2.6%).

Among monotherapy, 28 (24.36%) patients were on Sodium valproate monotherapy, 16 (13.92%) patients were on Carbamazepine monotherapy, 10 (8.7%) patients were on Phenobarbitone monotherapy and 3 (2.61%) patients had Phenytoin monotherapy and 1 patient was on Clobazam. 17 (14.78%) patients were on 3 or more drug which indicate that they are probably refractory seizures (table-21 & 22).

Table-22: Monotherapy

Monotherapy	**SVP**	**CBZ**	**PHT**	**PB**	**Others**
Patient n (%)	28 (24.36%)	16 (13.92%)	3 (2.61%)	10 (8.7%)	1 (0.9%)

SVP- sodium valproate, CBZ- Carbamazepine, PHT- Phenytoin, PB- Phenobarbitone

Person collecting drugs from hospital:

In our study 71 (61.74%) patient's mothers, 31 (26.96%) patient's fathers, 10 (8.69%) patient's other relatives and 3 (2.61%) patients themselves come regularly and collect antiepileptic drugs(AED) from our hospital.

Place of delivery and perinatal insult:

There was no statistical significant association between place of delivery and perinatal insult (P value-0.06). Even though we expect more perinatal insult in home delivery than institutional delivery it was not noted in our study (table-23).

Table-23: Place of delivery and perinatal insult

Place of delivery	MRI-PEC n (%)	MRI-others	Total	Chi square value-3.476 df-1	P value-0.06
Home delivery	6 (12.0%)	2 (3.10%)	8 (7.0%)		
Institutional delivery	44 (88.0%)	63 (96.9%)	107 (93.0%)		

Mode of delivery and perinatal insult:

50 patients in our study had evidence of hypoxic-hypoglycemic injuries. 38 of these patients were born of vaginal delivery and 12 patients were delivered by LSCS. The association between mode of delivery and perinatal injuries were not statistically significant (P value-0.939) (Table-24).

Table-24: Mode of delivery and perinatal insult

Mode of delivery	MRI-PEC n (%)	MRI-others	Total	Chi square value-0.006 df-1	P value-0.939
Vaginal delivery	38 (76.0%)	49 (75.40%)	87 (75.70%)		
LSCS delivery	12 (24.0%)	16 (96.9%)	28 (24.30%)		

Age of onset of seizure and perinatal encephaloclastic lesions:

In our study 58 (50.4%) patients had seizure onset in the first year of life (newborn and 1-12months). 31 (62.0%) of these patients had imaging evidence of perinatal injuries to brain. 19 (38.0%) out of 57 patients of later onset seizure (2^{nd} and 3^{rd} year) group had imaging evidence of perinatal injuries to brain. There is significant association between age of onset of seizure and imaging evidence of perinatal injuries to brain (P value 0.03). Earlier the onset of seizures more the possibility of imaging evidence of perinatal injuries (table-25).

Table-25: Onset of seizure and perinatal encephaloclastic (PEC) lesions

Seizure onset	**MRI-PEC n (%)**	**MRI-others**	**Total**	Chi square value-4.733 df-1	P value-0.03
1st year	31 (62.0%)	27 (41.50%)	58 (50.4%)		
Later(2^{nd}, 3^{rd} year)	19 (38.0%)	38 (58.5%)	57 (49.6%)		

Feeding and perinatal encephaloclastic (PEC) lesions:

Table-26: Feeding and perinatal encephaloclastic (PEC) lesions

Newborn feeding	MRI-PEC n (%)	MRI-others	Total	Chi square value-10.22 df-1	P value-0.001
Early(1-3hours)	25 (50.0%)	51 (78.5%)	76 (66%)		
Later	25 (50.0%)	14 (21.5%)	39 (34%)		

Totally 76 (66%) patients received early feeding (within 3 hours of birth) during the newborn period and 39 (34.0%) had late feeding (after 3 hours). There is a significant association between newborn feeding time and hypoxic-hypoglycemic injuries (P value-0.001). Late

feeding group had more number of patients with perinatal injuries in imaging (Table-26).

Cry and perinatal encephaloclastic (PEC) lesions:

Totally 31 patients had delayed cry at birth. 19 of these patients had evidence of perinatal injuries in imaging. 31 out of 84 normally cried patients had perinatal injuries. There is a significant association between delayed cry at birth and perinatal injuries in imaging (P value-0.02) (Table-27).

Table-27: Cry and perinatal encephaloclastic (PEC) lesions

Cry after birth	**MRI-PEC n (%)**	**MRI-others**	**Total**	Chi square value-5.48 df-1	P value-0.02
Normal	31 (62%)	53 (81.54%)	84 (73.04%)		
Delayed	19 (38%)	12 (18.46%)	31 (26.95%)		

Newborn admission and perinatal encephaloclastic (PEC) lesions:

30 out of 40 patients with newborn admission had perinatal injuries whereas only 20 out of 75 patients without newborn admission

had perinatal injuries. The association is statistically significant (P value-0.000) (Table-28).

Table-28: Newborn admission and perinatal encephaloclastic (PEC) lesions

Newborn admission	**MRI-PEC n (%)**	**MRI-others**	**Total**	Pearson Chi square value-24.80 df-1	P value-0.000
yes	30 (60.0%)	10 (15.40%)	40 (34.78%)		
No	20 (40.0%)	55 (86.60%)	75 (62.22%)		

Developmental delay and perinatal encephaloclastic (PEC) lesions:

Table-29: Developmental delay and perinatal (PEC) lesions

Development	**MRI-PEC n (%)**	**MRI-others**	**Total**	Pearson Chi square value-7.65 df-1	P value-0.01
Normal	29 (58.0%)	53 (81.50%)	82 (71.30%)		
Abnormal	21 (42.0%)	12 (18.50%)	33 (28.70%)		

The patients with developmental delay had perinatal injuries more commonly than patients with normal development. This is statistically significant (P value-0.01) (table-29).

Cognition and perinatal encephaloclastic (PEC) lesions:

Table-30: Cognition and perinatal encephaloclastic (PEC) lesions

Cognitive impairment	**MRI-PEC n (%)**	**MRI-others**	**Total**	Pearson Chi square value-20.36 df-1	P value-0.000
Present	35 (70.0%)	18 (27.70%)	53 (46.09%)		
Absent	15 (30.0%)	47 (72.30%)	62 (53.91%)		

The patients with cognitive impairment had perinatal injuries more commonly than patients with normal development. This is statistically significant (P value-0.000) (Table-30).

Focal epilepsy and perinatal encephaloclastic (PEC) lesions:

39 out of 63 patients with complex partial seizure of extra temporal origin had imaging evidence of perinatal injuries but only 9 out of 30 of patients with complex partial seizure of temporal origin had

perinatal injuries. This association is statistically significant (P value-0.004) (table-31).

Table-31: Focal epilepsy and perinatal encephaloclastic (PEC) lesions

CPS	**MRI- PEC n (%)**	**MRI-others**	**Total**	Pearson Chi square value-8.28 df-1	P value-0.004
CPS-T	9 (18.80%)	21 (46.70%)	30 (32.30%)		
CPS-ET	39(81.20%)	24 (53.30%)	63 (67.70%)		

AED and etiology:

Table-32: AED and perinatal encephaloclastic (PEC) lesions

AED	**MRI-PEC n (%)**	**MRI-others**	**Total**	Pearson Chi square value-9.56 df-1	P value-0.002
mono	17 (34.0%)	41 (63.10%)	58 (50.44%)		
poly	33 (66.0%)	24 (36.90%)	57 (49.56%)		

33 out of 57 patients with polytherapy had imaging of evidence of perinatal injuries whereas 17 out of 58 monotherapy patients had perinatal injuries. The association is statistically significant (P value-

0.004) (table-32). 44 of 87 vaginal delivery patients had monotherapy and 43 patients had polytherapy. 14 of 28 patients with LSCS delivery were on monotherapy and 14 patients had polytherapy. But there is no statistically significant association between mode of delivery and type of therapy (Pearson Chi Square value was 0.003 and P value was 0.96).

Sex and Birth weight:

Table-33: Sex and Birth weight

Sex	**Number**	**mean**	**S.D**	t-value	
Male	68	2.81	0.48	0.82	P value-0.40
Female	47	3.15	0.34	df-113	

The mean birth weights of males were 2.81kg and females were 3.15kg. However, the difference was not statistically significant (P value-0.40) (Table-33).

Perinatal encephaloclastic (PEC) lesions and Birth weight:

Patients with mean lower birth weight were significantly associated with imaging evidence of perinatal injuries than higher mean birth weight. The difference was statistically significant (P value-0.01) (Table-34).

Table-34: MRI perinatal encephaloclastic (PEC) lesions and Birth weight

MRI	Number	Mean wt.	S.D		
PEC	50	2.62	0.37	t-value-2.66 df-113	P value-0.01
MRI others	65	2.85	0.53		

Status epilepticus (SE) and imaging:

Table-35: Status epilepticus and perinatal encephaloclastic (PEC) lesions

Status (SE)	MRI-PEC n (%)	MRI-others	Total	Pearson Chi square value-0.47 df-1	P value-0.493
Yes	11(22.0%)	11 (16.90%)	22 (19.1%)		
No	39(78.0%)	54 (83.10%)	93(80.9%)		

In our study, 22 patients had status epilepticus in the past, 11(50%) of them had hypoxic -hypoglycemic injuries in MRI brain, 1(4.5%) patient had mesial temporal sclerosis and 10(45.5%) patients had normal imaging. The association was not statistically significant (P value-0.49).

DISCUSSION

DISCUSSION

In our study 115 patients with seizure onset in the first three years of their life were included.

Demography and familial factors:

The youngest patient was one year old and the oldest patient was 36 years of age. The mean age of the study population was 11.4 ± 7.58 years. There were 68 (59.10%) males and 47 (40.90%) females with male: female ratio of 1.4:1 and this is similar to a study done by V.Udani et al (M: F-1.8:1). Epilepsy is slightly more common in males than in females but the difference is not statistically significant.[39] Sex of the patient probably did not affect the seizure prognosis.[33]The mean age of onset of epilepsy was 14.8 ± 11.2 months. There were 21 (18.26%) patients with seizure onset in the newborn period and 38 (33%) patients had seizure onset between 1 to 12 months of age. 51.25% of patients had onset of seizure in the 1st year of their life. This is in concordance with other studies showing the incidence of epilepsy is high in the infantile population.[4,39,45] The mean age of onset of seizure was 13.9 months in V.Udani et al., study which is comparable with our study.[5]

As per updated Kuppusamy's scale (2007),[67] 111 (96.50%) patients belong to class-4 socio economical status and 4 (3.50%) patients belong to class-3 category. Some of the studies have documented that epilepsy is more common among children living in low socio economical condition, irrespective of their ethnicity which is true in our study also.[39,41] But however as our hospital is a Government hospital which treats for the poorer section of the society free of cost, the high incidence of class-4 category could be due to the above factor. 80% of our study population were from urban area and 20% were from rural area. This may be because our hospital is located in urban area and people from rural area have to travel a long distance during epileptic emergencies and to collect drugs.

In our study, 18 patients were aged 18 years and above but only 3 of them have completed secondary school. This is consistent with the fact that patients with epilepsy may be dissuaded from completing school education as well due to the cognitive disturbances associated with epilepsy.[68]

Consanguinity was found in 28.7% of our study population. A study from Kerala has noted 13.5% of paternal consanguinity in children with epilepsy. Frequency of consanguinity in India varies from 15.9% to 32.9 %.[43] South India, including Tamilnadu comes under high

prevalence zone (>20%) of consanguinity except for the state of Kerala.[43, 69] Most recent study from Chennai concluded that paternal consanguinity was 37% among mentally retarded children.[70] This may be the reason for the high rate of consanguinity found in our study population.

Family history of seizure was present in 33% of our patients, 31.6% in another study.[61] Three other studies had documented much lower rates: 24.1%, 13.7%, and 22.2% respectively.[45,47,48] However its association with development of epilepsy was significant in other studies by Monetti VC et al ., and in a study from Kerala.[57,58]

Maternal factors and perinatal factors:

Recurrent abortion was present in 18 (15.7%) mothers of our patients, eclampsia in 2 patients, and gestational diabetes in 3 and antepartum bleeding in 1 mother of our patients. Most of the studies have however found that these factors not associated with development of epilepsy.[43,51,53,57] In one study alone, vaginal bleeding had significant association for the development of epilepsy.[61]

In our study, the institutional delivery was 93% and home delivery was 7%. As per the government of India report, the institutional deliveries in Tamil nadu is more than 90% and it is 100% in

Chennai.[71,72,73] In our study, 75.7% of deliveries were normal vaginal delivery and 24.3% deliveries were LSCS delivery. WHO recommended maximum rate of LSCS was 15%. But, 36.2% of LSCS delivery were documented in a study from Italy and 45% of LSCS delivery were noted in a study from Chennai, India.[74,75] As per the study done by Ayhan Sucaket et al., emergency LSCS has increased the maternal and fetal mortality than elective LSCS delivery.[76] LSCS delivery may adversely affect breast feeding.[75] However the association between mode of delivery (LSCS or vaginal) and perinatal injuries were not statistically significant (P value-0.939) in our study. Even though the institutional delivery was very high, 43.48% of our cohorts suffered perinatal insult. 88% of these perinatal insults were noted among institutional delivery group. There was no statistically significant association between place of delivery and perinatal insult (P value-0.06). Even though we expect more significant difference in perinatal insult between home delivery and institutional delivery it was not noted in our study. The reason for this could be due to the fact the level of perinatal care is more important determinant factor than the mode and place of delivery. Neonatal hypoglycemic brain injuries (NHBI) were significantly associated with LSCS deliveries in a study by V,Udany etal.[5] However no certain

reasons for the same could be explained. One reason perhaps could be a higher rate of emergency LSCS deliveries in tertiary care hospitals.

A study done by Sarad Kumar Singh et al,[77] concluded that even though hospital delivery has increased significantly in India, the expected decline in perinatal mortality rate (PNMR) was not present. He also emphasized that improving the quality of care in institutional delivery (perinatal care) is vital for reduction of PNMR. Recently, Government of India has started newborn survival program (Navjaat Shishu Shurakha karyakaram) to train health care personal for newborn care to tackle this type of problems.[77]

20.9% of our cohorts had birth weight less than 2.5 kg and 34.0% of patient received newborn feeding later than 6 hours of birth. Patients with lower mean birth weight had perinatal injuries more than higher mean birth weight group. The difference was statistically significant (P value-0.01).There is significant association between newborn feeding time and hypoxic-hypoglycemic injuries (P value-0.001). Late feeding group had more number of patients with perinatal injuries. Similar observation was also found in V.Udani et al., study.[5]

27% of our cohort had delayed cry at birth, 34.8% had admission during newborn period and newborn seizure was noted in 18.3% of

patients. There was a significant association between delayed cry at birth and perinatal injuries in imaging (P value-0.02). Patients with newborn admission had significant perinatal injuries than patients without newborn admission. The association is statistically significant (P value-0.000). In one study delayed cry and newborn admission was found to have significant association with epilepsy onset in univariate analysis but not on multivariate analysis.[43] Javad Akhondian et al., found that newborn seizure was 17.6% in children with intractable epilepsy similar to our observation.[47] Francesco Pisani et al., noted that moderate HIE with or without newborn seizure was not significantly related to post neonatal epilepsy but severe HIE has.[78]

Most of the studies from developed nations did not find any causal relationship between maternal or early neonatal factors and risk for development of epilepsy.[52,53,57] However, most of the studies from developing countries found these factors to be still relevant.[5,43,46,51] This may be due to the reason the etiology of early onset epilepsy is largely different in developed versus developing countries.

Clinical factors:

In our cohort, 33 (28.7%) patients had delayed developmental milestones, 53 (46.1%) patients had cognitive impairment, and 10

(8.7%) patients had psychosis. 2 (1.7%) patients had facial dysmorphism and 2 other patients had neurocutaneous markers. 10 (8.7%) patients had focal neurological deficits with spasticity and 5 patients had microcephaly and one patient had Fahr's disease. One patient had features of tuberous sclerosis. One patient's mother had Gilbert syndrome and one patient's father had thalassemia.

The patients with developmental delay had more perinatal injuries than patients with normal development. This is statistically significant (P value-0.01). The patients with cognitive impairment had more perinatal injuries than patients with normal development. This is statistically significant (P value-0.000). The presence of developmental delay; cognitive impairment and psychosis were well documented in literatures which are in concordance with our study.[5,44,48,79,80,81] More than 75% of children with epilepsy had developmental delay on routine screening by a research group.[79] Age of onset of seizures, frequency and duration of epilepsy are the determinants of cognitive impairment in epilepsy.[80] Post ictal psychosis includes various psychotic disorders which are associated with poorly controlled epilepsies and it has to be recognized and treated.[81] Focal neurological deficit, microcephaly were also observed in other studies.[5,47] Children with neonatal hypoglycemic brain injuries (NHBI) tend to have more number of microcephaly,

mental retardation and epilepsy and lower rate of spasticity and dystonia.[5] A similar trend was noted in our study.

Epilepsy characters:

71.3% of our cases had single type of seizure, 25.2% had 2 seizure types and 3.5% of patients had 3 seizure types. A study from The Cleveland clinic foundation noted single seizure type in 53% of their cohorts, two seizure types in 42% of cases, three seizure type in 3% of cases and four seizure type in 3% of cases.[66]

Our cohorts had hypermotor seizures (tonic, clonic, myoclonic, epileptic spasms, atonic, versive and unclassified motor seizures) in 87%, hypomotor seizures in 9.6% and automotor motor seizures in 3.5%. Among the hypermotor seizure, 48 (41.7%) patients had clonic seizures; tonic seizures were present in 23 (20%) patients, 13 (11.3%) patients had epileptic spasms, 6 (5.2%) patients had atonic seizures, versive seizures were present in 4 (3.5%) patients, unclassified motor seizures in 5 (4.3%) patients and one patient had myoclonic seizure.

The Cleveland clinic study noted 79% of motor seizures, 20% of hypomotor seizures and 1% of automotor seizure.[66] Mild variation in semiological classification in our study may be due to the reason that we classified the seizures purely on the basis of carefully elicited clinical

history rather than ictal Video EEG. Tuqba Hirfanoqlu et al., has done a study of seizure semiological classification based on history from patients and based on video EEG. He compared the consistency of history based classification with video EEG classification. He concluded that the semiological seizure classification based on history can reliably be used in day to day outpatient visits.[82]

93 (80.9 %) of our cases were having focal epilepsies, 21 (18.3 %) cases had generalized epilepsy and gelastic seizure in 1 (0.9%). In the focal epilepsy group, Complex partial seizure of extra temporal was more commonly observed (54.8%) than temporal origin (19.14%). Generalized seizures were noted in 18.3% of our cases. Dura-Travel T et al., have documented 52.9% focal epilepsy, 43.6% generalized epilepsy and 3.5% had epilepsy with an undermined localization in his study.[83] The higher incidence of generalized epilepsy in the later study could be due to the fact that the age of onset of epilepsy included in the study was up to 15 years.

In our study, 39 out of 63 patients with complex partial seizure of extra temporal origin (CPS ET) had imaging of evidence of perinatal injuries but only 9 out of 30 patients with complex partial seizure of temporal origin (CPS T) had perinatal injuries. This association is

statistically significant (P value-0.004). Most of the perinatal insults were found at the occipital and parieto- occipital regions. This may be the reason for predominance of CPS ET in our study.

There is a 2.6% reduction in weekly seizure frequency after treatment, 7.8% reduction in monthly seizure frequency and 27 (23.5%) patients did not have seizures during last one year. The seizure frequency was increased by 4.3% at 3months and 8.3% at 6 months respectively. Even though 23.5% of patients had seizure control, majority continue to have seizures with lesser frequency, which may suggest that a true remission was not seen among majority of our patients.

22 (19.14%) patients had history of status epilepticus (SE) before entering into regular treatment at our centre. In our study 11 of these status epilepticus patients had perinatal injuries in imaging and other 11 patients had normal imaging. But there was no statistically significant association between imaging etiology and status epilepticus (P value-0.49). There was no similar study for us to make a comparison in this regard.

Imaging characters and Etiology by imaging:

In our study, 54 (46.96%) patients had normal MRI Brain (non lesional) and 61 patients had abnormal (lesional) imaging. 50 (43.5%) patients had perinatal encephaloclastic conditions (HIE, NHBI, PVL), mesial temporal sclerosis were found in 4 (3.5%) patients, heterotopias were noted in 2 (1.7%), focal cortical dysplasia in 1 (0.9%), hypothalamic hamartoma in 1 patient, cortical tuber in 1 patient, metachromatic leukodystrophy in 1 patient and Fahr's disease in 1 patient (Table-17).

Parieto-occipital and occipital region were the common sites of perinatal injuries. Some authors had documented parieto-occipital and occipital region were commonly involved in NHBI (neonatal Hypoglycemic injuries).[5] In our study, perinatal encephaloclastic conditions (included hypoxic ischemic encephalopathy, neonatal hypoglycemic injuries, periventricular leucomalacia, and focal infarcts) were found in 50 (43.5%) patients. Whereas only 11(9.5%) patients had other etiology (table-17).This observation is in concordance with other studies.[5,62,84-90] Studies from developed countries found that cortical dysplasia, agyria-pachygyria and tuberous sclerosis are the main

etiology for remote symptomatic epilepsy occurring in infancy and early childhood in their population.[51,91]

In this study 53% of patients were having symptomatic epilepsies and 47% of patients having non lesional epilepsies. This observation is much different from V. Udani et al., study in which 83 out of 100 cases were diagnosed to have symptomatic epilepsies and 17 cases were non lesional.[5] Probably, higher tesla MRI may give more information about these non lesional epilepsies in future.

In countries like India, perinatal factors are still play a major role in the causation of infantile and early childhood onset remote symptomatic epilepsy. As per the WHO/ILAE estimation, almost 10% of incidental epilepsy can potentially be prevented. Birth asphyxia and neonatal hypoglycemia often coexist and causes severe brain damage.[5] Hence, increasing the institutional deliveries and improving the quality of care during high risk period (pre and perinatal) will go a long way in reducing the perinatal hypoxic- hypoglycemic brain injuries in babies, which will have direct effect in reducing early onset remote symptomatic epilepsy in our part of the country.

Electroencephalography:

18 (15.65%) patients had focal interictal epileptiform discharges (IED), 15 (13.04%) patients had multifocal interictal epileptiform discharges (IED) and 82 (71.31%) did not have interictal epileptiform discharges (IED). Further uncertainty as regards to semiological localisation and lateralisation as well as ictal and interictal electrographic data need to be resolved by long term Video EEG monitoring.

Antiepileptic treatment (AED):

58 (50.4%) patients were on single anti epileptic drug (monotherapy) and 57 (49.6%) patients were receiving 2 or more anti epileptic drugs (polytherapy). 40 (34.8%) patients were on 2 drugs, 12 (10.4%) patients were taking 3 drugs, 4 (3.5%) patients were on 4 drugs and 1 (0.9%) patient was on 5 drugs.

Sodium valproate was the most commonly used monotherapy (24.36%) in our study, followed by carbamazepine (13.92%) and phenobarbitone (8.7%). 49.60% of our patients receiving polytherapy, of which 17 (14.78%) were receiving 3 or more drugs. In patients with 2 drug group, the 2^{nd} drug is being titrated to its maximum tolerable dose hence the true refractoriness could not be assessed at present. However,

we can reasonably conclude that a significant number of our patients ultimately may end up in drug refractory state in near future.

The total annual cost of epilepsy (direct + indirect) in India was INR 13,755, as per a study done by S. V. Thomas et al, during 1998.[91]The direct cost was INR 3,728 and indirect cost was INR 10,030. If we apply the above total cost to our study population the total cost the management of epilepsy would be INR 13,755 per person per year. It would be still higher if we consider the today's market rate. This estimate indicates the economical burden of epilepsy on the state, apart from its negative impact on patients and their families.

The limitation of our study is that it is based on patients attending a tertiary care hospital they may not be truly representative of the community sample.

SUMMARY
AND
CONCLUSION

SUMMARY AND CONCLUSION

- Our study empathetically establishes the contribution of perinatal encephaloclastic conditions particularly hypoxic-hypoglycemic injuries as an important cause of the early childhood onset epilepsies.

- Mode of delivery and Institutional delivery did not have the expected impact in reducing perinatal hypoxic-hypoglycemic brain injuries and it emphasises the need to improve quality perinatal care among institutional deliveries in our country.

- Delayed cry at birth, newborn admission, low birth weight, delayed initiation of newborn feeding are the significant risk factors for perinatal encephaloclastic conditions.

- Focal epilepsy, particularly focal epilepsy of extra temporal origin is the commonest epilepsy type observed.

- In addition to perinatal encephaloclastic conditions (43.5%) and other symptomatic epilepsies (9.5%), 47% of patients had non lesional epilepsies.

- Nearly 50% of patients were on polytherapy.

In developing countries like India, the perinatal factors still play a major role in the causation of infantile and early childhood onset remote symptomatic epilepsy. Perinatal hypoxia and undiagnosed neonatal hypoglycemia are potentially preventable risk factors for the development of the same. Hence quality perinatal care may be the need of the hour to prevent early onset childhood epilepsy in a large group of patients.

Further care of these patients, may also need comprehensive epilepsy care units for optimal management of epilepsy and related issues in these patients in tertiary care hospitals. These above measures would help to alleviate the morbidity and socio-economic burden associated with epilepsy in these patients.

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ANNEXURE

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Clinical and etiological Profile of Epilepsy with onset In the First three years of life in a tertiary care Hospital

Principal Investigator : Dr. A.Rajendran

Designation : PG in DM (Neuro)

Department : Department of Neurology
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 06.03.2012 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

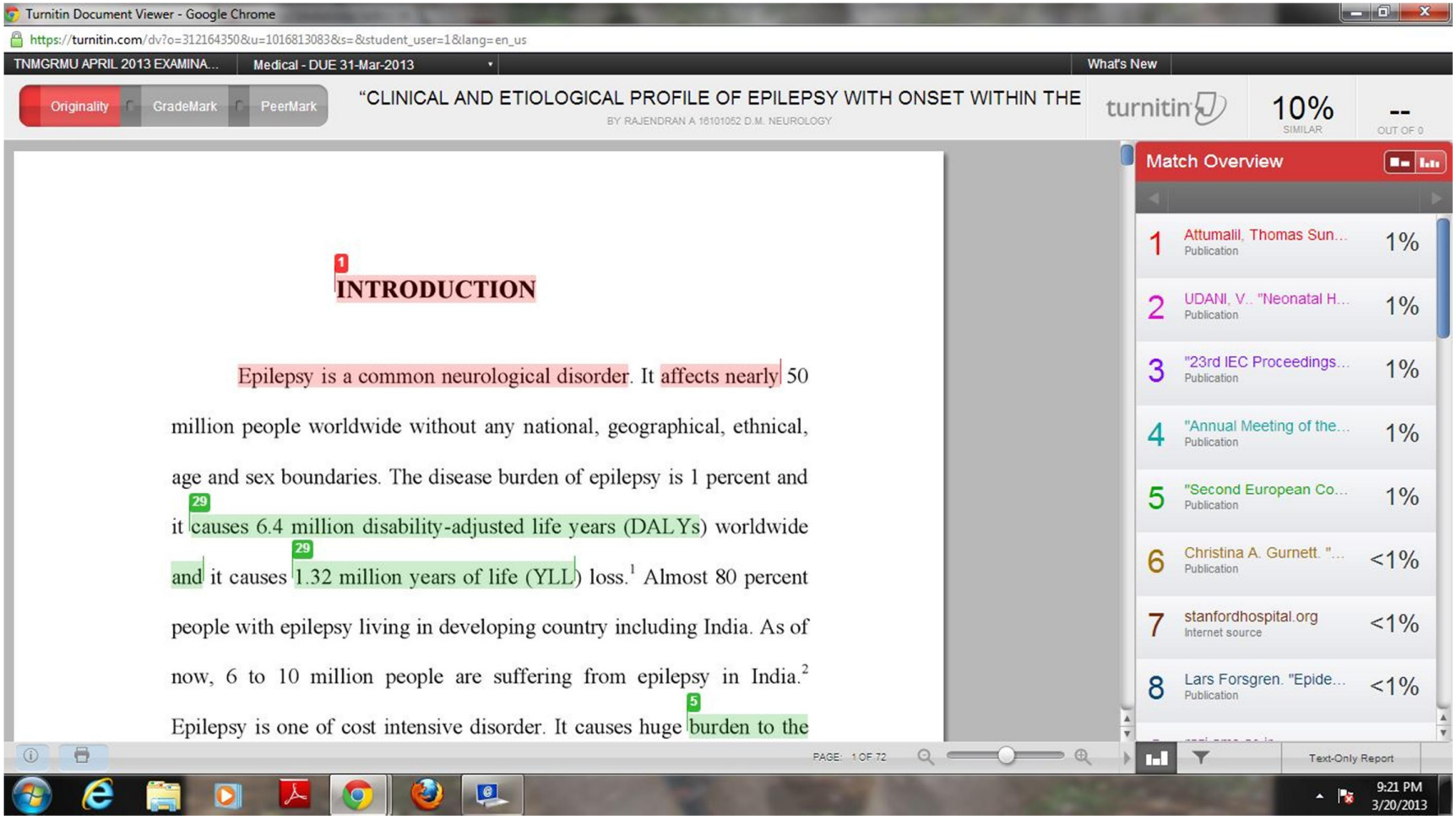
The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

19/2/13

MEMBER SECRETARY,
IEC, SMC, CHENNAI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INTRODUCTION
Epilepsy is a common neurological disorder. It affects nearly 50 million people worldwide without any national, geographical, ethnical, age and sex boundaries. The disease burden of epilepsy is 1 percent and it causes 6.4 million disability-adjusted life years (DALYs) worldwide and it causes 1.32 million years of life (YLL) loss.[1] Almost 80 percent people with epilepsy living in developing country including India. As of now, 6 to 10 million people are suffering from epilepsy in India.[2] Epilepsy is one of cost intensive disorder. It causes huge burden to the
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E-mail	rajendranmddm@gmail.com
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INTRODUCTION Epilepsy is a common neurological disorder. It affects nearly 50 million people worldwide without any national, geographical, ethnical, age and sex boundaries. The disease burden of epilepsy is 1 percent and it causes 6.4 million disability-adjusted life years (DALYs) worldwide and it causes 1.32 million years of life (YLL) loss.1 Almost 80 percent people with epilepsy living in developing country including India. As of now, 6 to 10 million people are suffering from epilepsy in India.2 Epilepsy is one of cost intensive disorder. It causes huge burden to the individuals, health care providers and society at large.3 The first year of human life is associated with the highest...

PROFORMA

Name; Age; Sex;

Age of onset of seizures;

Address; Study no/ neuro no.

Marital status; Residence;

Socio-economical status;

Literacy status;

Consanguinity; Degree;

Family h/o seizures; relation to index case;

Family h/o febrile seizure; relation to index case;

Mother – antenatal registration;

High risk factors;

Torch infections PIH/ Eclampsia GDM/DM APH/PPH Drug intake;

H/o recurrent abortion (spontaneous) before the index case;

Medical illness; other specify;

Natal history;

Place of delivery; 1) home 2) Institutional

Institution; medical college/ G.H/ PHC/ Sub centre/ private

Mode of delivery;

Normal vaginal delivery/ assisted delivery/operative (LSCS) / others specify.

IF LSCS; indication

Person conducted delivery; Trained / untrained person

Trained person- Dr / Staff nurses/ VHN/ANM

Neonatal history;

Apgar score;

Baby looks; normal / pale / blue

Cry after birth; normal cry / delayed cry

H/o neonatal resuscitation detail;

Newborn Feeding history; hour of feeding stared immediate/ 1hour/2 hour/3huor/4hour/5/ 6hour, later etc.,

Type of feeding; breast feeding / prelactial feeding/ artificial feeding/ intravenous/ppn/tpn/ others detail.

H/o newborn seizure;

If yes; Time/ day of onset of seizure;

Altered level of consciousness in newborn period; yes/no

Newborn fever; yes/no

Documented Hypoglycemia; yes/ no relevant details

Birth weight;

Gestational Age; preterm / term /post term

Newborn Admission details;

Developmental history;

Gross motor

Fine motor

Language

Social / behavior

H/o antecedental events meningo encephalitis/ head injuries/ vaccine related / others specify.

Age of onset of first seizures;

Habitual seizures;

Aura type; detailed description;

Ictal semiology detailed description;

Tonic / clonic / myoclonic / atonic / versive / epileptic spasm / hypomotor/ Automotor / unclassified motor/ Automotor

Seizure type; SPS / CPS/ CPS with secondary generalization/ generalized.

Duration of each attack;

Tongue bite / urination;

Post ictal confusion/ post ictal weakness

No. of seizure type; one/ two/ three/ four/ more;

Seizure free period between 1st seizurs and habitual seizures;

Triggers if any;

Clusters;

Nocturnal seizures only / day alone / day and seizures;

Status epilepticus history;

Non convulsive status;

Frequency of seizure before regular treatment; detailed description

Frequency of seizure before regular treatment; detailed description

Cognitive/ behavior changes;

Psychosis;

Educational difficulty;

Others;

Present medication;

Name of the drug dose in mg.

No. of drugs

Seizure episodes on medication;

Drug compliance detailed description;

Adverse effect of drug;

Person collecting drug from hospital;

Treatment of co morbid conditions;

Immunization history;

Examination;

Ht Wt HC: Handedness;

Micro / microcephaly

Facial dimorphism / neurocutaneous markers details;

Eye signs; cataract / strabismus/ microcornea and others

Fundus; optic atrophy, cherry red spot, chorio-retinitis and others

Mental retardation / cognitive impairment;

Focal neurological deficit; Detail;

Others;

MRI brain;

Interictal EEG;

Other investigations;

Follow up;

Medical events and others

நோயாளி தகவல் தாள்

மூன்று வயதிற்குள் ஏற்பட்ட வலிப்பு வியாதியுடன் உள்ள வலிப்பு நோயாளிகளிடம் காணப்படும் தன்மைகளும், வலிப்பு நோயின் காரணங்களும் என்ற ஆய்விற்கு - ஒப்புதல் படிவம்

நோயாளிகளுக்கான தகவல்: ஆராய்ச்சியின் நோக்கமும், பயன்களும்.

உங்கள் பங்கேற்பு திட்டமிடப்பட்டுள்ள இந்த மருத்துவ ஆராய்ச்சி ஆய்வின் நோக்கம்:

மூளையில் ஏற்படும் - முரண்பாடுகளுடன் கூடிய அதிகப்படியான அல்லது ஒருமித்த நரம்புகளில் செயல்பாடுகளால் வலிப்பு வியாதி ஏற்படுகிறது. இது அனைத்து வயதினரையும் பாதிக்கிறது. குழந்தைகளுக்கும், அதிக வயதானவர்களும் வலிப்பு வியாதி அதிகளவில் ஏற்படுகிறது.

குழந்தைகள் பிறப்பின் போது ஏற்படும் பாதிப்புகளாலும், மூளை காய்ச்சல், முளை வளர்ச்சியில் ஏற்படும் குறைபாடுகளாலும் மற்றும் சில வேளைகளில் உடலில் சர்க்கரை, தனிமவகைகள் குறைவதாலும் வலிப்பு நோய் ஏற்படலாம்.

இந்த ஆய்வில் - முதல் மூன்று வயத்திற்குள் ஏற்பட்ட வலிப்பு வியாதியுடன் உள்ள நோயாளியிடம் காணப்படும் காரணங்கள் மற்றும் தன்மைகள் குறித்து ஆய்வு மேற்கொள்ளவதன் மூலம் இது போன்ற வலிப்பு வியாதியுடன் உள்ளகளுக்கு மேம்பட்ட சிகிச்சை கிடைக்கவும், தாய்சேய் நல சிகிச்சை மேம்பாட்டினால் இதுபோன்ற வலிப்புகள் குறைய வாய்ப்பு உள்ளதா? என தெரிந்து கொள்ளவும் வாய்ப்பு ஏற்படும்.

ஆய்வு நடைமுறைகள்

மூன்று வயதிற்குள் ஏற்பட்ட வலிப்பு வியாதியுடன் உள்ள வலிப்பு நோயாளிகள் மட்டுமே இந்த சிகிச்சையில் சேர்த்துக் கொள்ளப்படுவார்கள். இந்த பரிசோதனையானது 12 மாதங்கள் நடைபெறும்.

அந்தரங்கத்தன்மை

உங்கள் / உங்கள் பிள்ளையின் / உறவினரின் மருத்துவப் பதிவேடுகள் மிகவும் அந்தரங்கமாக வைத்துக் கொள்ளப்படும் மற்றும் மற்ற பிற மருத்துவர்கள் / விஞ்ஞானிகள் / இந்த ஆய்வின் தணிக்கையாளர்கள் அல்லது ஆராய்ச்சி ஆதரவாளர்களின் பிரதிநிதிகள் ஆகியோரிடமும் அவை வெளிப்படுத்தப்படும். இந்த ஆய்வின் முடிவுகள் அறிவியல் பத்திரிக்கைகளில் பிரசுரிக்கப்படலாம். ஆனால், பெயரை வெளியிடுவது மூலம் நோயாளிகள் அடையாளம் காட்டப்பட மாட்டார்கள்.

ஆய்வில் பங்கேற்கும் நோயாளியின் கடமைப் பொறுப்புகள்

உங்களை / உங்கள் உறவினரை கவனித்துக்கொள்ளும் மருத்துவருடன் நீங்கள் முழுமையாக ஒத்துழைக்க வேண்டும் மற்றும் உங்கள் மருத்துவரால் குறிப்பிடப்படும் மருத்துகளை தர அனுமதிக்க வேண்டும் என்று உங்களைக் கேட்டுக்கொள்றோம். சிகிச்சையளிக்கும் மருத்துவர் அளிக்கும் அறிவுரைகளை பின்பற்ற வேண்டும் என்றும், என்னென்ன செய்ய வேண்டும், என்னென்ன செய்யக்கூடாது என்று உங்களிடம் கூறப்பட்டுள்ளவற்றிலிருந்து சற்றும் விலகக்கூடாது என்றும் நீங்கள் எதிர்பார்க்கப்படுகிறீர்கள்.

ஆய்வில் உங்கள் பங்கேற்பு மற்றும் உங்கள் உரிமைகள்

இந்த ஆய்வில் உங்கள் / உங்கள் உறவினரின் பற்கேற்பு தன்னிச்சையானது மற்றும் காரணங்கள் எதையும் கூறாமலேயே நீங்கள் / உங்கள் உறவினர் இந்த ஆய்விலிருந்து எந்த ஒரு நேரத்திலும் விலகிக் கொள்ளலாம். எப்படியிருந்தாலும், உங்கள் / உங்கள் உறவினரின் உடல் நிலைக்கேற்ப உங்களுக்கு / உங்கள் உறவினருக்கு பொருத்தமான சிகிச்சை அளிக்கப்படும். ஆய்வில் பங்கேற்க நீங்கள் மறுப்பதால், அடுத்து வரும் ஆராய்ச்சி ஆய்வுகளில் உங்கள் / உங்கள் உறவினர் பங்கேற்பை மறுப்பது போன்ற எந்த வித அபராதமும் விதிக்கப்படாது. உங்களை / உங்கள் உறவினரை கவனித்துக் கொள்ளும் மருத்துவருடன் முழுமையாக ஒத்துழைக்க நீங்கள் சம்மதிக்க வேண்டும். எந்த ஒரு நேரத்திலும், நீங்கள் மோசமாக உணர்ந்தாலோ அல்லது வேறு ஏதேனும் உடல் நலக்குறைவு உண்டானாலோ, தயவு செய்து, உங்களை / உங்கள் உறவினரை கவனித்து வரும் மருத்துவரிடம் உடனடியாக தெரிவிக்கவும். சிகிச்சை உங்களுக்குப் பொருத்தமாக இருக்காது என்று தோன்றினால் உடனடியாக நிறுத்தப்படும். உங்கள் சம்மதம் இன்றியே கூட ஆய்வு நிறுத்தப்படுவது சாத்தியமே.

வேறு ஏதேனும் கேள்விகள் / பிரச்சினைகள் பற்றி நீங்கள் கேட்க விரும்பினால், கீழ்க்கண்ட நபரைத் தொடர்பு கொள்ளவும்.

தனியாகப் பிரிதெடுத்து, ஆய்வில் பங்கேற்பவரிடம் தரப்பட வேண்டும்.

நோயாளி தகவல் தாள்

மூன்று வயதிற்குள் ஏற்பட்ட வலிப்பு வியாதியுடன் உள்ள வலிப்பு நோயாளிகளிடம் காணப்படும் தன்மைகளும், வலிப்பு நோயின் காரணங்களும் என்ற ஆய்விற்கு - ஒப்புதல் படிவம்

நோயாளியின் பெயர் ..... பாலினம் ஆண் ..... பெண் .....

வயது ..... வருடங்கள் அல்லது பிறந்த தேதி .....

நோயாளியை தொடர்பு கொள்ளும் முகவரி .....

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நோயாளியின் தொலைபேசி எண்.

நோயாளியின் தந்தை / கணவர் / உறவினர் பெயர் .....

		பங்கேற்பவரின் கையொப்பம்/பெரு விரல் பதிப்பு
1)	மேல் குறிப்பிடப்பட்டுள்ள ஆய்வின் ..... தேதியிட்ட நோயாளிகளுக்கான செய்தி நான் படித்திருக்கிறேன் மற்றும் புரிந்திருக்கிறேன்/ விவரிக்கப்பட்டுள்ளேன். கேள்விகள் கேட்கவும் அனுமதி வழங்கப்பட்டுள்ளேன் என நான் உறுதி செய்கிறேன்.	
2)	இந்த ஆய்வில் பங்கேற்பது என் / என் உறவினரின் சொந்த விருப்பப்படியே என நான் அறிந்திருக்கிறேன். மேலும் என் / என் உறவினரின் மருத்துவ சிகிச்சை கவனிப்பு அல்லது சட்ட பூர்வ உரிமைகளுக்கு பாதிப்பு ஏற்படாமல் நான் எந்த நேரத்திலும் விலகிக் கொள்ளலாம் என்பதை அறிந்திருக்கிறேன்.	
3)	எத்திக்ஸ் கமிட்டி மற்றும் ரெகுலேட்டரி அதாரிட்டீஸ்க்கும் நான் இந்த ஆய்விலிருந்து விலகினாலும் தற்போதைய மற்றும் எதிர்கால இந்த ஆய்வு சார்ந்த என் / என் உறவினர் உடல்நல குறிப்புகளை என் அனுமதியின்றி பார்க்க முடியும் என நான் அறிகிறேன்.	
4)	இந்த ஆய்வில் கிடைக்கப்பெறும் குறிப்புகள் மற்றும் முடிவுகளை உபயோகப்படுத்த தடை செய்ய மாட்டேன் என சம்மதிக்கிறேன். ஆனால் அவைகள் விஞ்ஞானம். ஆராய்ச்சி கட்டுரைகள் போன்ற சம்மந்தப்பட்டவைகளுக்கு பயன் உள்ளதாக இருக்க வேண்டும்.	
5)	மேற்கூறிய ஆய்வில் பங்கேற்க நான் சம்மதிக்கிறேன்.	

ஆய்வில் பங்கேற்பவர் / சட்டபூர்வமாக
ஏற்கப்பட்ட நபர் கையொப்பம் அல்லது
பெருவிரல் பதிவு

(2)

சுய ஒப்புதல் படிவம்

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி மருத்துவமனை, சென்னை - 600 001.

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் எண் :

பங்கு பெறுவர் இதனை (✓) குறிக்கவும்.

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும். அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் / என் உறவினர் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் / என் உறவினர் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் / என் உறவினர் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்த கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணிக்கு தெரிவிப்பேன் என உறுதி அளிக்கிறேன். ☐

இந்த ஆய்வில் எனக்கு இரத்தம், சிறுநீர், எக்ஸரே, ஸ்கேன் (MRI Scan), E.E.G. உட்பட அனைத்து பரிசோதனைகனையும் செய்து கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின்/உறவினரின் கையொப்பம் ..... இடம் ..... தேதி
கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர் .....

MASTER CHART - 1 A

SL. NO.	NAME	AGE	SEX , MALE -1, FEMALE-2	MARITAL STATUS, UNMARRIED -1, MARRIED -2	SE SCALE CLASS - 3, CLASS 4	RESIDENCE, URBAN -1, RURAL-2	LITERACY STATUS, IL-1, PS-2, SS-3, C-4, DROP OUT-5, NA-6	CONSANGUINE-OUS Y-1,N-2	FAMILY H Y-1,N-2	REGISTERE D Y-1,N-2	TORCH -1, PIH-2, DM/GDM -3, APH/PPH-4, DRUG INTAKE -5	BOH-1, REC ABORT-2	PLACE OF DEL , HOME-1, INST-2	INST DEL TYPE SUBTYPES-mc-1, gh-2, phc-3, subcen-4, pvt-5	MODE OF DEL N-1, ASS-2, LSCS-3	LSCS- IND GIVE CODES-rptls/cpd-1, fd-2, obstru-3,oligo-4,Breech-5,Pih/Eccl ampsia-6	PERSON COND DEL, TRAINED-1, UNTRAINED-2	CRY AFTER BIRTH, YES-1, N-2, NOT KNOWN -3	FEDDING START 0-3:1, 3-6:2, LATER :3	TYPE OF FEED-pre lac/af-1, bf-2, ivf/ppn/tp n-3	H/O NEW BORN SZ Y-1,N-2	BIRTH WT, 1,2,3,4	GEST AGE 1-PRE,2-TERM,3-POST	NEW BORN AD Y-1, N-2	DEV. Normal-1, Abnormal-2	AGE OF ONSET OF SZ new born-1, 1st year,2 2nd y-3, 3rd y,4	AURA1,2, 3,4,no-5,other6	SPS-1,CPS-2,CPS WITH SEC-3, GEN-4, Gelastic seizure-5	SL. NO.	CPS-T :1, CPS-ET:2	FREQ once in w-1, m-2, 3months-3, 6months-4, year-5, occationally-6
1	Ramesh	18	1	1	4	1	3	2	2	1		1	2	3	1		1	1	1	2	2	2	2	2	1	2	2	1	1	3
2	Gajalakshmi	16	2	1	4	1	3	2	1	1	4		2	1	1		1	2	3	3	2	2	2	1	1	4	5	3	2	1	3
3	Vaidegi	10	2	1	4	1	3	2	1	1		2	2	3	1		1	1	1	2	2	2	2	2	2	2	1	2	3	2	6
4	Purnima	17	2	1	4	1	3	2	1	1			1		1		2	2	3	1	2	1	1	2	2	4	5	2	4	2	6
5	Suresh	22	1	1	4	2	3	1	1	1		2	2	3	1		1	1	1	2	2	1	2	2	2	3	5	2	5	2	5
6	Abdul rahman	18	1	1	4	1	2	2	2	1			2	1	1		1	1	1	2	2	2	2	2	1	3	5	2	6	2	4
7	Anu	6	2	1	4	2	2	1	2	1		2	2	1	3	1	1	1	3	3	2	2	2	1	2	3	5	2	7	2	5
8	Yuvanesh	2	1	1	4	2	6	2	2	1		2	2	1	3	3	1	2	3	3	1	2	2	1	2	1	5	2	8	2	5
9	Moulana	18	1	1	4	1	5	1	2	1		2	2	2	1		1	2	2	2	2	2	2	2	1	3	5	2	9	2	1
10	Harishankar14	14	1	1	4	1	3	2	2	1		2	2	2	1	3	3	1	2	3	3	1	2	2	1	1	1	5	2	10	2	5
11	Kamalesh	3	1	1	4	2	6	2	2	1			2	3	1		1	2	1	2	2	3	2	2	1	2	5	4	11		1
12	Gopalakrishnan	14	1	1	4	1	5	2	2	1			2	2	1		1	2	3	3	1	1	2	1	2	1	5	2	12	2	2
13	Gowtham	16	1	1	4	1	5	2	2	1			2	1	3	2	1	2	1	2	2	2	2	2	2	3	5	4	13		5
14	Jack fernando	5	1	1	3	1	6	2	2	1			2	2	1		1	1	3	3	1	3	2	1	1	1	5	2	14	2	5
15	Vishal	5	1	1	4	1	6	2	2	1			2	1	1		1	1	1	2	2	2	2	2	1	4	5	2	15	1	2
16	kurushith	15	1	1	4	1	5	1	2	1			2	1	3	1	1	1	1	2	2	2	2	2	1	2	5	2	16	2	6
17	Gowsalya	14	1	1	4	2	2	2	2	1			2	3	1		1	1	1	2	2	2	2	2	1	2	5	2	17	2	6
18	Ganesamoorthy	16	1	1	4	1	2	2	2	1			2	2	1		1	2	3	3	2	2	2	1	1	3	5	2	18	1	6
19	Pushparaj	13	1	1	4	2	5	1	1	1			2	1	1		1	1	1	2	2	2	2	2	1	2	6	2	19	1	6
20	Devaraj	35	1	1	4	1	3	1	2	1			2	4	1		1	1	1	2	2	2	2	2	1	2	1	2	20	1	6
21	harish	9	1	1	4	1	2	2	2	1			2	5	1		1	1	1	2	2	4	2	2	1	3	6	2	21	1	1
22	pavivardhini	7	2	1	4	2	2	1	1	1			2	5	1		1	1	1	2	2	2	2	1	1	4	3	1,3	22	2	2
23	pavithra	10	2	1	4	2	2	2	2	1			2	2	1		1	2	3	3	1	2	2	1	2	1	5	2	23	2	6
24	Albert	29	1	1	4	1	2	2	2	1		1	2	1	1		1	1	1	2	2	2	2	1	1	2	5	2	24	2	6
25	Theneka	4.5	2	2	4	1	6	1	2	1			2	1	1		1	1	1	2	2	2	2	2	2	3	5	4	25		5
26	Louis roshan	4	1	1	4	1	6	2	1	1			2	1	1		1	1	1	2	2	2	2	2	1	2	5	3	26	2	2
27	Mohamed imthiyas	12	1	1	4	1	3	2	1	1			2	5	1		1	1	3	2	2	3	2	2	2	3	3	2	27	2	6
28	Renilda vincy	8	2	1	4	1	2	2	2	1			2	4	1		1	1	1	2	2	3	2	2	1	2	5	4	28		6
29	Blessy	8	2	2	4	2	2	1	2	1			2	5	3	1	1	1	3	1	2	2	2	2	1	3	5	2	29	1	6
30	Gunasekar	6	1	1	4	1	2	2	1	1		2	2	3	1		1	1	1	2	2	2	2	2	1	4	5	2	30	2	6
31	Arthy	12	2	1	4	2	3	1	2	1			2	2	1		1	1	1	2	2	2	2	2	1	3	5	2	31	2	3
32	Daniel	4.5	1	1	4	2	6	2	2	1			2	1	3	3	1	1	1	2	2	3	2	2	1	4	5	3	32	2	6
33	Muthu meena	4.5	2	1	4	1	6	2	2	1			2	2	3	3	1	2	3	3	1	2	2	1	1	1	5	2	33	2	4
34	Sowmiya	13	2	1	4	1	3	1	1	1			2	1	1		1	2	3	3	2	2	2	1	1	2	5	4	34		1
35	Kaviya	11	2	1	4	1	3	1	1	1			2	1	1		1	1	1	2	2	1	2	2	1	3	5	4	35		1
36	Fariza	7	2	1	4	1	2	2	2	1			2	1	3	1	1	1	1	2	2	2	2	2	1	4	5	4	36		6
37	Tamilarasi	16	2	1	4	2	1	2	2	1			2	3	1		1	1	1	2	2	2	2	2	1	2	5	2	37	1	3
38	Karan	10	1	1	3	1	2	2	1	1			2	1	3	1	1	1	1	2	2	3	2	2	2	2	5	3	38	2	2
39	Rajeswari	7	2	1	4	1	2	2	2	1	3		2	1	1		1	1	1	2	2	1	2	2	1	4	5	4	39		6
40	Nazir hussain	6	1	1	4	1	1	2	2	1			2	5	1		1	1	1	2	2	2	2	2	1	4	5	4	40		6
41	Emmanuel	12	1	1	4	1	1	2	2	1	2		2	1	1		1	2	3	1	2	1	1	1	2	4	5	3	41	2	6
42	Anish	2	1	1	4	1	6	2	1	1			2	3	1		1	2	1	2	2	2	2	2	1	2	5	4	42		2
43	Kavitha	11	2	1	4	1	3	2	2	1		2	2	1	1		1	1	1	2	2	2	2	2	1	3	5	3	43	2	3
44	Govinesh	1	1	1	4	1	6	2	2	1			2	1	1		1	1	3	3	2	1	1	1	1	2	5	4	44		1
45	Sabana	8	2	1	4	1	2	2	2	1			2	1	1		1	1	1	2	2	1	2	2	1	3	5	3	45	1	6
46	Sowmiya	2	2	1	4	1	6	1	2	1			2	1	1		1	1	1	2	2	2	2	2	1	2	5	3	46	2	2
47	Gowtham	8	1	1	4	1	2	2	2	1			2	1	1		1	1	1	2	2	1	2	2	1	2	5	3	47	2	6
48	Nareshkumar	2.5	1	1	4	1	6	2	2	1			2	1	1		1	1	1	2	2	1	1	1	2	3	5	3	48	2	1
49	Rushiya	1	2	1	4	2	6	2	1	1			2	5	3	2	1	2	3	3	1	1	2	1	2	1	5	3	49	2	1
50	Saran	8	1	1	4	1	2	2	2	1		2	2	1	3	1	1	1	2	2	1	2	2	1	2	1	5	3	50	2	3
51	menaka	4	2	1	4	1	6	2	2	1			2	1	3	5	1	1	1	2	2	2	2	2	1	3	5	3	51	1	6
52	Mohan Raj	8	1	1	4	1	2	2	2	1		2	2	1	3	1	1	1	1	2	2	2	2	2	1	3	5	4	52		6
53	Sridhar	10	1	1	4	1	2	2	1	1			2	1	3	3	1	1	1	2	2	2	2	2	1	3	5	2	53	2	6
54	Jenita	11	2	1	4	1	2	1	2	1			2	2	1		1	1	1	2	2	2	2	2	1	4	5	4	54		6

MASTER CHART - 2A

55	Venkatesh	8	1	1	4	1	1	2	2	1			2	1	1		1	1	1	2	2	2	2	2	1	2	5	3	55	1	6
56	ragul	8	1	1	4	2	2	2	2	1			2	1	3	1	1	1	1	2	2	2	2	2	2	4	5	5	56		1
57	Aravind	13	1	1	4	1	3	2	1	1			1		1		2	1	1	2	2	2	2	2	1	4	5	2	57	2	3
58	Tansila Fathima	3.5	2	1	4	1	6	2	2	1	3		2	2	3	1	1	1	1	2	2	2	2	2	2	2	5	2	58	2	1
59	Babu	7	1	1	4	1	2	2	1	1			2	4	1		1	1	1	2	2	2	2	2	1	2	5	2	59	1	4

60	Nandhini	11	2	1	4	1	3	1	1	1			2	2	1		1	1	1	2	2	2	2	2	1	4	5	2	60	2	6
61	Sindu	22	2	1	3	1	3	2	2	1		2	2	5	1		1	2	2	1	1	2	2	1	1	1	5	2	61	2	3
62	Janani	4	2	1	4	1	6	2	1	1	3		2	1	3	2	1	1	1	2	2	1	2	1	2	2	5	2	62	2	4
63	Harish kumar	3	1	1	4	1	6	2	1	1			2	2	3	2	1	1	1	2	2	1	2	2	1	4	5	2	63	2	2
64	Ajith	15	1	1	4	2	3	1	2	1		2	2	5		1	1	2	2	3	1	2	2	1	1	1	5	2	64	2	1
65	Velmani	19	1	1	4	1	5	2	1	1			2	1	1		1	1	1	1	2	1	2	2	2	3	5	3	65	2	2
66	Akash	15	1	1	4	1	3	2	1	1			2	1	1		1	1	1	2	2	2	2	2	1	2	5	2	66	1	2
67	Sangeetha	27	2	1	4	2	3	2	2	1			2	3	1		1	1	1	2	2	2	2	2	2	3	5	3	67	1	2
68	Renuga Devi	13	2	1	4	1	3	2	2	1	2		2	5	3	6	1	1	1	3	2	2	2	2	1	4	5	2	68	2	3
69	Deepak	10	1	1	4	2	1	2	2	1			2	5	1		1	1	1	2	2	2	2	2	1	2	5	2	69	1	1
70	Prakash	15	1	2	4	1	2	1	1	1			2	5	1		1	2	2	1	2	2	2	2	1	2	5	2	70	2	2
71	Lalitha	35	2	2	4	1	2	2	1	1			1		1		2	1	1	1	2	2	2	2	1	4	5	2	71	2	6
72	Hemanth kumar	13	1	1	4	1	3	1	2	1			2	1	1		1	1	1	2	2	2	2	2	1	2	5	3	72	2	6
73	Sekar	14	1	1	4	1	3	1	2	1			2	1	3	1	1	1	1	2	2	2	2	2	1	4	5	2	73	2	3
74	Parthiban	14	1	1	4	1	1	1	1	1		2	1		1		2	1	3	1	2	1	2	2	2	2	5	3	74	1	6
75	Kathirvel	3.5	1	1	4	1	6	2	2	1		2	2	2	1		1	1	1	2	2	2	2	2	1	4	5	3	75	1	6
76	Chandru	7	1	1	4	1	2	2	2	1			2	1	3	1	1	1	1	2	2	2	2	2	1	4	5	2	76	1	5
77	sundar	22	1	1	4	1	3	1	1	1		2	2	3	1		1	2	3	1	2	2	2	1	1	2	5	3	77	2	6
78	Chithra	22	2	1	4	1	3	1	2	1			1		1		2	2	2	3	1	1	2	1	2	1	5	3	78	2	2
79	Anitha	25	2	2	4	2	3	2	1	1			2	1	1		1	1	3	3	1	1	2	1	2	1	3	3	79	2	2
80	Sivakumar	16	1	1	4	1	1	2	2	1			2	3	1		1	2	3	3	1	2	2	1	2	1	5	2	80	2	4
81	Santhosh kumar	6	1	1	4	2	2	1	2	1			2	5	1		1	2	3	3	2	2	2	1	1	3	5	2	81	2	6
82	Saranya	18	2	1	4	1	3	1	2	1			2	5	1		1	1	1	2	2	2	2	2	1	3	5	3	82	1	6
83	Naren Karthik	8	1	1	4	1	2	2	2	1			2	2	1		1	1	1	2	2	2	2	2	1	2	5	2	83	1	3
84	Sadhana	3.5	2	1	4	1	6	1	1	1			2	2	1		1	1	1	2	2	2	2	2	1	4	5	3	84	1	3
85	Sujatha	4	2	1	4	1	6	1	2	1			2	3	1		1	2	1	2	2	2	2	2	1	3	5	3	85	1	2
86	Keerthana	6	2	1	4	1	2	2	2	1		2	2	5	1		1	1	1	2	2	2	2	2	1	3	5	3	86	2	2
87	Omprakash	5	1	1	4	1	2	2	2	1			2	1	1		1	2	3	3	2	2	2	1	2	4	5	4	87		6
88	Nishawthini	3.5	2	1	4	1	2	1	1	1			2	1	1		1	1	3	3	2	2	2	1	1	4	5	4	88		4
89	Vidhya	6	2	1	4	1	2	2	2	1			2	1	3	1	1	1	3	1	2	2	2	2	2	3	5	2	89	2	4
90	Sivasankari	8	2	1	4	1	2	2	1	1			2	1	1		1	1	1	2	1	2	2	1	1	1	5	2	90	2	2
91	Divya	17	2	1	4	2	3	2	2	2			2	2	1		1	1	1	2	2	2	2	2	1	3	5	3	91	1	2
92	Pradeep kumar	14	1	1	4	1	1	2	2	1			2	3	1		1	1	3	3	2	2	2	2	2	2	5	3	92	1	5
93	Vajitha Begam	19	2	1	3	2	4	2	1	1			2	1	1		1	1	1	1	2	1	2	2	1	2	5	2	93	1	1
94	Srinivasan	26	1	1	4	1	1	1	2	1			2	1	1		1	2	2	3	1	2	2	1	2	1	5	2	94	1	4
95	Gowrishankar	14	1	1	4	1	3	2	2	1			2	1	1		1	2	1	3	2	2	2	2	2	2	5	2	95	2	6
96	Barathi	2	2	1	4	1	6	2	1	1			2	2	3	1	1	1	1	2	2	1	2	2	1	2	5	4	96		3
97	Mohammed ali	16	1	1	4	2	3	2	2	1		2	2	3	1		1	1	1	2	2	2	2	2	1	2	3	3	97	2	6
98	Ganesan	17	1	1	4	1	3	2	2	1			2	1	3	2	1	2	3	3	1	1	2	1	2	1	5	3	98	2	5
99	Anwar basha	8	1	1	4	1	2	2	2	1			2	4	1		1	1	1	2	2	3	2	2	1	4	5	1	99	2	6
100	Ram shri	3	2	1	4	1	6	1	1	1			2	4	1		1	1	1	2	2	2	2	2	1	2	5	4	100		6
101	Karthikeyan	13	1	1	4	1	3	2	1	1			2	3	1		1	2	2	3	2	2	2	1	1	4	3	2	101	2	1
102	Alex	9	1	1	4	1	2	2	2	1			2	1	3	1	1	1	1	2	2	2	2	1	1	3	5	3	102	2	5
103	Nagaraj	16	1	1	4	1	3	1	2	1		2	1		1		2	1	1	1	1	2	2	1	1	1	5	3	103	2	3
104	Sudharson	3	1	1	4	1	6	2	2	1			2	2	3	2	1	1	1	1	2	2	2	2	1	2	5	4	104		2
105	Mercy	4	2	1	4	1	6	2	1	1			2	2	1		1	1	1	2	2	2	2	2	1	2	5	2	105	1	3
106	Nowsath ali	6	1	1	4	1	6	1	2	1			2	1	1		1	2	3	3	2	2	2	1	1	3	5	4	106		3
107	Aravind	9	1	1	4	1	2	2	1	1			2	1	1		1	2	3	3	1	2	2	1	1	1	5	2	107	1	6
108	Ranjitha	4	2	1	4	1	6	2	1	1			2	5	1		1	1	1	2	1	1	2	1	1	1	5	4	108		6
109	Buvaneshvari	7	2	1	4	1	2	2	2	1			2	1	1		1	1	3	3	1	1	2	1	1	1	1	2	109	2	2
110	Vijayakumar	36	1	1	4	1	1	2	2	1			1		1		2	2	3	1	1	2	2	1	2	1	5	3	110	2	6
111	Akalya	7	2	2	4	1	2	2	2	1			2	1	1		1	1	1	1	2	2	2	2	1	2	5	4	111		6
112	Gopinathan	29	1	1	4	2	3	1	1	1			2	5	1		1	1	1	2	2	1	2	2	1	4	5	3	112	1	1
113	Amina	8	2	1	4	1	3	2	2	1			2	1	1		1	1	1	2	2	2	2	2	1	4	1	2	113	2	5
114	Sivaprakash	15	1	1	4	1	1	1	2	1			2	1	1		1	1	1	2	2	2	1	1	2	3	5	2	114	2	3
115	Raja	25	1	1	4	1	1	2	2	1			1		1		2	1	3	3	2	1	2	1	2	2	5	2	115	2	6

MASTER CHART - 1 B

CLUSTER Y-1,N-2	NOCTURNAL -night-1, day-2, both-3	STATUS Y-1,N-2	COG IMP 1-PRESENT, 2-ABSENT	PSYCHOSES 1-PRESENT, 2- ABSENT	SZ FREQUENCY on AED-once in w-1, m-2, 3months-3, 6months-4, year-5, nil-6	SZ FREE PERIOD-<1y-1, 1-5y-2,5-10-3,>10y-4,nil-5	AED , MONO-1/POLYTHERAPY-2	POLYTHERAPY - NO.OF DRUGS	PERSON COLLECTING DRUG, SELF-1, OTHERS-2	FACIAL DYSMORPHISM PRESENT-1, ABSENT-2	NEUROCUTANEOUS MARKERS, PRESENT-1, ABSENT-2	FOCAL DEFICITS , PRESENT-1, ABSENT-2	CPS-TEMPORAL-1, EXTRATEMPORAL-2	EEG - 1-focal IED, 2-multifocal IED, 3-Generalised, 4-normal	MRI BRAIN,GLIOSIS-1, FCD-2, heteritropia-3, MTS-4, normal-5 , Fahr's-6,cortical tubers-7,hypothalamic hamartoma-8, mld-9	occip-1, p/o-2,perirol-3, periro-p/o-4 ,all-5, b/l periventri-6,fron-parietal-7,Frontal-8,Parietal-9,Cerebellum-10	drugs-Pht-<400/d-1, 400-600-2, 600-800-3,>800-4	cbz-<400/d-1, 400-600-2, 600-800-3,>800-4	svp-<400/d-1, 400-600-2, 600-800-3,>800-4	pb-<30/d-1,30-60-2, 60-90-3,>90-4	clonazipam-y-1 n-2	clobazam y-1, n2	drugs others-y1n2	name of drug	seizure type; T-1, M-2,C-3,A-4,V-5,ES-6,H-7,AUT-8,UNC-9	Semiology : S-1,TWO-2,THREE-3	H/O NEW BORN SZ Y-1,N-2	AGE OF ONSET OF SZ 1,2,3,4	weight in Kg	age of onset of seizures in months
2	2	2	2	2	3	5	2	3	2	2	2	2	1	4	5			2	1	2					6	1	2	2	3	6
2	2	1	1	1	6	5	2	2	2	2	2	2	1	4	1	9			3						diazepam	1	3	2	4	2.75	30
2	2	1	1	2	1	2	1		2	2	2	2	2	4	1	2		2								3	1	2	2	2.6	8
2	2	2	1	2	1	3	1		2	2	2	2	2	4	1	2		3								1	1	2	4	2.6	28
2	2	2	1	1	1	1	1		2	2	2	2	2	4	5				1							1	1	2	3	2.3	18
2	1	2	2	2	6	2	1		2	2	2	2	2	4	5		1									3	1	2	3	2.8	16
2	2	2	1	2	2	2	1		2	2	2	2	2	4	5					2						6	1	2	3	3	22
2	2	2	1	2	1	1	1		2	2	2	1	2	4	1	2				2						3	1	1	1	3.05	1
2	2	2	1	2	4	5	2	2	2	2	2	2	2	4	1	1	1	1								3	2	2	3	2.6	13
2	2	2	1	2	6	4	2	2	2	2	2	2	2	4	1	5		1			1					7	2	1	1	2.7	1
1	3	2	1	2	3	5	2	5	2	2	2	2		4	1	2		1	1	2	1					3	1	2	2	3.7	3
2	2	2	2	2	3	2	2	2	2	2	2	2	2	4	1	2		2	2							3	2	1	1	2.2	1
2	3	2	1	2	5	5	2	2	2	2	2	2		4	5				1				1	Diazepam		3	1	2	3	2.7	18
2	1	2	2	2	6	5	1		2	2	2	2	2	1	1	3			1							3	1	1	1	3.4	24
1	3	2	2	2	3	5	2	2	2	2	2	2	1	1	1	5		1	1							7	1	2	4	2.6	30
2	2	2	2	2	5	4	2	2	2	2	2	2	2	1	1	5		1	2							1	2	2	2	2.7	4
2	3	2	2	2	5	3	1		2	2	2	2	2	4	5			2								1	2	2	2	2.9	18
2	2	2	2	2	5	3	1		2	2	2	2	1	4	2				1							5	1	2	3	2.75	24
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MASTER CHART - 2 B

2	3	2	2	2	4	2	1		2	2	2	2	1	4	5				2						7	2	2	2	3.25	11
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